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RADIATION HAZARDS OF DIAGNOSTIC X RAYS.[1]

By ALICE STEWART,
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SINCE it has now been placed beyond doubt that ionizing radiations can cause leukæmia or cancer in man as well as in animals, it should be obvious that studies of the carcinogenic effects of diagnostic radiology are not made to prove this point. Their interest lies in the fact that they provide a convenient—indeed, the only—way of discovering whether very small doses of ionizing radiations have this effect in man. By definition, such investigations must be based on human data, and they are therefore bound to take the form, not of planned experiments, but of what are nowadays known as medical surveys or, more precisely, social medicine surveys. Such investigations can be designed to demonstrate the existence of factors which alter the prevalence of diseases and their distribution—so-called risk factors—but are quite unsuited to studying the factors which are directly responsible for a particular disease taking a particular form—so-called specific causes of disease.

At this point it is, perhaps, worth while drawing attention to the fact that when one is considering what it is that causes a disease to appear, it is important to remember that there are both specific and non-specific causes. Upon the former will depend the pathological and clinical manifestations of the disease, and upon the latter the number of cases. By specific causes we mean such things as bacteria, viruses, vitamins and hormones, and proof that they are responsible for a characteristic set of clinical and pathological manifestations depends on tests which are biologically specific. (The tests required are listed in Koch's postulates.) The non-specific causes include such things as age, sex, occupation, habits and locality, and because their effects are medically non-specific they cannot be tested in this way. They demand instead investigations of the type that I am about to describe—that is to say, investigations whose end-point is a demonstration of statistical relationships between presumed risk factors and presumed medical consequences. It is, of course, true that the

[1] Read at the North Queensland Medical Conference, Mackay, September 22 to 29, 1958.

discovery of a causal relationship in this way may eventually lead to the discovery of the specific cause of the disease; but final proof can never be established within the framework of the original investigation.

I have developed this theme at some length, because it is my experience that the present state of confusion about what to expect from social medicine research is caused by a failure to distinguish clearly between what may be called the qualitative aspects of disease, which are the main concern of laboratory and clinical research workers, and the quantitative aspects of disease, which are the subject-matter of social medicine research. Practising clinicians are necessarily interested in both aspects; but just as it has been left to laboratory and hospital workers to isolate the specific causes of disease and to observe their effects, so it has been left to social medicine research workers to isolate non-specific causes and measure their effects.

Recent studies of the carcinogenic effects of diagnostic radiology provide an excellent example of this type of work, which always follows the set pattern of (i) formulating a working hypothesis based on one or more arbitrary assumptions; (ii) testing this assumption by collecting standardized data from test and control groups and systematically examining these data for evidence of statistically significant relationships. In the present case, the arbitrary assumption that we made was that exposure to small, apparently harmless doses of ionizing radiations imperceptibly increases the risk of leukæmia or cancer. This was subseequntly justified: first, by our discovery that in a large group of children who had died of leukæmia or cancer before the age of 10 years, there were twice as many who had been exposed to diagnostic X-rays before birth as in a control group; and secondly, by the work of an independent observer in America, who repeated the investigation and obtained virtually the same results.

It is important to realize that this is all that has been done, and that so far there has been no question of anyone's setting out to prove that the development of leukæmia or cancer in any given individual is a direct consequence of exposure to diagnostic X-rays. As has already been stated, proof of a causal relationship of this kind requires totally different methods of investigation, and is appropriate only if previous work has already indicated the nature of the causal factor.

I hope by this time that I have made it clear that the findings of the Oxford survey of malignant disease in childhood are not comparable with the findings of a laboratory investigation. They can, however, be compared with what it is possible to learn by taking the facts recorded in official statistics of mortality and subjecting them to detailed cross-analysis. Needless to say, the object of this type of "desk-work" is never to discover the cause of a disease — this may or may not be known before the figures are examined — but to show how the numerical frequency of the disease is influenced by such things as age, sex, occupation and locality.

It is, for instance, possible to discover from official statistics of mortality that deaths from pulmonary tuberculosis are consistently more frequent among people who work in factories than among people who work on the land—a fact which makes it a reasonable proposition to base an investigation on the assumption that cross infection at work is one of the factors affecting the distribution of this disease. This particular assumption is justified because we already know that the specific cause of pulmonary tuberculosis is a bacillus which is air-borne and has infective properties. When the official vital statistics relating to diseases of unknown ætiology, such as leukæmia or cancer, are examined, there is less chance of their indicating a specific line of inquiry. On the other hand, the mere fact that the specific cause is not known makes a detailed examination of this type of data particularly worth while, for in such circumstances a display of the death rates for the diseases at different times and in different circumstances may promote fruitful ideas which it would be hard to come by in other ways.

It was, for instance, a detailed consideration of the death rates for lung cancer in different decades and in different parts of Great Britain which first justified a detailed study of the disease in relation to smoking habits. The idea had been floating around in people's minds for some time; but it was not until Dr. P. Stocks (working on figures supplied by the General Register Office) showed that the death rates for lung cancer were wholly consistent with such an idea that the Medical Research Council decided to support a large-scale survey.

The Oxford survey of malignant disease in childhood is no exception to this rule, and was, in fact, based on a detailed analysis of official vital statistics relating to leukæmia by my statistical colleague, Mr. David Hewitt. It is therefore appropriate to preface the findings of the childhood survey with some of his findings. Hewitt was not, of course, the first person to notice that the death rate for leukæmia was rising, but he did draw attention to the differences between different countries and the differences between different groups in the same country. Thus, in the United States the death rate for the white population was found to be approximately twice as high as that for the coloured population; in the British Isles, the rate for England and Wales was found to be 50% lower than the high rate in the United States, but considerably higher than the rate for the Irish Republic. Within England and Wales the rates were higher for high-income groups than for low-income groups, higher in the north than in the south, and higher for males than for females. Hewitt made three other important observations. First, by dint of making a cohort analysis of deaths, he showed that the rise in the death rate had affected people born during the present century more than people who were born in the nineteenth century; secondly, the rise was particularly marked for children aged under five years, both in this country and in the U.S.A.; finally, the death rates for these children were remarkable, inasmuch as there was a peak of mortality between the ages of two and four years, and this peak had become sharper with the passage of time. After considerable deliberation, we — that is to say, Hewitt, my other colleague, Dr. J. W. Webb, and myself—decided to put the following interpretation on these findings.

First, we thought it reasonable to assume that whatever was causing the rise in the leukæmia death rate had probably been operating throughout the present century, and was probably affecting children at least as much as, and possibly more than, adults. (This particular interpretation of the figures was supported by a later analysis of deaths for all forms of cancer, which showed that during the last 20 years the increase in death rate for all forms of cancer combined had been far greater for children than for adults.) We also decided that if we ever had a chance to make a survey, we would be wise to include among possible risk factors diagnostic radiology, first because it had been increasingly used since the beginning of the century, and secondly because it was probably used more often by well-to-do persons than by poor persons. Finally, we decided in the first instance to base our survey on children rather than on adults, partly because it seemed to us that it would be far easier to reconstruct the life histories of children than of grown-ups, and partly because of the peculiar peak of mortality at the ages of two to four years. This feature of the death rates also reminded us that it would be important not to forget that a child's history begins, not when it is born, but when it is conceived.

The principal objection to restricting the survey to children was, of course, the rarity of the disease in this age group; however, weighed against this was one important advantage. For obvious reasons, the facts that we required could not be collected direct from the children concerned, but must be obtained from responsible adults. It was, therefore, not essential for us to base our proposed investigation on live patients with leukæmia and cancer. This had the practical advantage that, provided the General Register Office agreed to cooperate, we might be able to discover the relatives of all the

children who had died of these diseases. How many of these children it might be possible to include in the survey would have to be decided later; but it was clearly desirable to know about them all, because in the whole of the country the total number of deaths from leukæmia in children aged under 10 years had been running at less than 300 *per annum*, and the corresponding number for other forms of cancer was not much larger.

Needless to say, it is not an easy matter for a very small department to organize a survey on a very large scale. On the other hand, we knew that our public health service possessed an elaborate system of welfare services for mothers and children, and that medical officers of health took a special pride in doing all they could to promote the health of the children in their locality. Provided, therefore, that some of these doctors could be persuaded to do the field work for us, and provided that we could work out a scheme for collecting a uniform set of records from different areas, we might not be faced with an impossible task.

In the event, every single one of the 196 local health authority departments in England, Wales and Scotland agreed to help by following up all the cases in their areas, and in this way all the children who had died of leukæmia or cancer during the three years 1953-1955 were included in the survey. For obvious reasons, not all these children appear in our final report. A few mothers refused to be interviewed, a few general practitioners said that it would not be wise to approach the parents, and several families had moved to unknown addresses. We also decided that where it had not been possible for the same doctor to investigate a case and the corresponding control, we would exclude the records from the final analysis. Nevertheless, out of a total of just over 1600 available cases, 1299 were included.

Besides interviewing the mothers of these children, each public health doctor who took part in the survey agreed to interview a control mother who had a child of the same age and sex as the corresponding subject and had been picked at random from a local birth register. In this way we obtained a control group which, if not ideal, was at least better than one consisting of children attending hospitals or welfare clinics, children who, from the point of view of social class, health supervision and sickness rates, are surprisingly different from the population at large.

There is unfortunately not time to discuss in detail the elaborate procedures which were undertaken to remove what the statisticians call "unknown bias" from the control group. These are described in detail in the report we have published, and if you have read this I hope you will agree that everything possible was done to show that the principal finding of the survey—namely, that pre-natal exposures to X rays increase the risk of a child's subsequently dying of leukæmia or cancer—is not an artefact. An important reason for our feeling more confident on this score than we might otherwise have been is that already a second survey, based on the original Oxford one, has been carried out in America and found to support this conclusion. On this occasion the facts were obtained, not from the mothers of dead and live children, but direct from doctors' records; thus the one weak link in our own chain of evidence was strengthened.

As has already been stated, the principal finding of the childhood survey was the discovery that at a particular age a particular type of diagnostic X-ray examination carries with it a small, but definite, carcinogenic risk. There were other findings—as, for instance, that first-born children and children whose mothers were aged over 40 years when they were born, are more likely to die of leukæmia than other children; that two diseases—mongolism and leukæmia—are linked in a peculiar way; that recovery from acute pulmonary infection is occasionally followed by the development of leukæmia; and finally that exposures to X rays in infancy may also carry a leukogenic risk. This is all that I have time to tell you about the childhood survey, but I thought you might also be interested to know that we have already embarked on a survey of adult leukæmia. The object of this investigation is to discover whether there is any association between previous occupations, illnesses and X-ray examinations, and the subsequent development of leukæmia. In order to carry out this survey, we have thought it necessary to interview patients with leukæmia before they die, and to match the records obtained in this way with similar records from patients with cancer and from healthy people. This has meant devising a system of live notifications and making arrangements for the leukæmia and cancer patients to be interviewed before they die. For this reason, there is no question of the second survey's covering the whole country. However, a large number of pathologists have agreed to cooperate and are notifying us of the cases that come their way. In addition, most medical officers of health who helped with the first survey have again agreed to interview the patients and controls. We hope eventually to include over 1000 cases of leukæmia and an equivalent number of cases of other cancers and controls, matched for age, sex and locality in the same way as in the childhood survey; but at the time of writing the total number of survey trios for which a full complement of data is available is only 408. These include 58 cases of acute leukæmia, 165 cases of chronic or subacute lymphocytic leukæmia, 156 cases of chronic or subacute myeloid leukæmia, and 29 cases of lymphosarcoma.

Thus far, only the results of a preliminary analysis of the X-ray histories are available. The general impression obtained from these figures is one of remarkable similarity between the three groups. There are, however, two categories which are tending to concentrate in the leukæmia series—namely, the one which includes 20 or more diagnostic X-ray examinations of the limbs (with no additional radiotherapy), and the one which includes radiotherapy directed towards a thoracic or abdominal organ. If these findings are confirmed in a larger series, we shall have seriously to consider the possibility that even in adults gene mutations are not the only consequences of exposing the trunk to repeated small doses of X rays.

ILLUSTRATIONS AND REPORTS OF LATE EFFECTS OF RADIATION.[1]

By A. G. S. COOPER,
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IN recent years a great deal of attention has been focused on the interesting and important subject of radiation hazards. The practising radiotherapist has been aware for many years that his invisible life-saving ray is also a noxious agent in greater or lesser degree, and has stressed this fact in pressing for a full training of medical graduates entering the specialty. Recent research, as we have just heard, serves to emphasize this conception, and demonstrates that there are sequelæ even more unpleasant than was first anticipated. The radiational effects to be described fall into the following three broad categories: (i) tissue effects, including hæmic changes (*a*) in patients, (*b*) in medical and industrial radiation workers, (*c*) in personnel exposed accidentally and in atomic warfare; (ii) (*a*) effects on the fœtus of irradiation to the mother, (*b*) leukæmia as a sequel to exposure to radiation; (iii) genetic hazards.

Tissue Effects.

In this medical audience, a knowledge of the present scope, achievements and commoner late effects of radiotherapy can be taken for granted. For instance, the frequently seen late radiation effects, such as the

[1] Read at the North Queensland Medical Conference, Mackay, September 22 to 29, 1958. This paper was illustrated by slides, many in colour, too numerous for reproduction.

atrophic or telangiectatic scar, need no special mention. In tissues which have received heavy local irradiation, the usual late findings are atrophy of the skin and fibrosis of underlying soft tissues, an effect which is often unavoidable in the treatment of advanced lesions. There may be sudden breakdown and necrosis of tissues, specially in cartilage or bone. The development of carcinoma in epithelial tissue and of sarcoma in tissues of mesodermal origin is an occasional finding. The differential absorption in bone of X rays in the superficial and deep therapy ranges was not fully appreciated until some twelve years ago, when it was ably reported by Spiers (1946).

Cataracts and severe anæmias have followed irradiation. Both cancer of the thyroid gland and leukæmia in childhood have been sequelæ to the irradiation of the enlarged thymus gland in infancy.

Microcephaly has been reported as an occasional finding after irradiation with large doses to the fœtus in utero. Examples occurred in pregnant women survivors at Hiroshima.

The successful irradiation of a malignant tumour hinges on the therapeutic ratio, which expresses the relationship between tumour lethal dose and tissue tolerance. This is illustrated by the finding that a large range of malignant tumours can be given a cancericidal exposure of radiation which is less than the accepted tolerance of normal tissues. It is important to realize that a cutaneous malignant tumour developing in the treatment site after an interval of ten years or more may be a cancer induced by radiation. It will be agreed that if this happened in 2% of patients surviving after treatment of a malignant lesion, it would not constitute a reason for the selection in the first instance of a less suitable alternative method of treatment. It would seldom be justified, however, to apply radiotherapy carrying this type of hazard to a benign lesion in a young subject.

An example claimed as an exception to this rule is the case of A., examined at the age of one month suffering from multiple hæmangiomata of the face, neck and shoulders, with extension to the larynx requiring tracheotomy. Superficial X-ray therapy to the surface lesions and deep X-ray therapy to the larynx were administered as life-saving measures. The dosage given was approximately 1700*r* at a half-value layer of four millimetres of aluminium over a period of six months. Twelve years later, the child was treated for several basal cell carcinomata of the skin in the shoulder and neck scars and for hypothyroidism.

Patient B., now aged 11 years, was treated in infancy in Melbourne for angiomata on the left side of the face and the left auricle. The treatment given consisted of four doses of 600*r* and one of 750*r*, a total of 3150*r*, over a period of 12 months ending in October, 1947. A small ulcer developed in the scaphoid fossa of the left ear in October, 1955, and when it was removed in November, 1956, it proved to be a basal cell carcinoma.

Master C., aged six and a half years, presented on January 29, 1958. His parent stated that he had developed a painless swelling on the right side of his face and neck ten weeks previously, which had steadily enlarged. Examination of the child revealed a massive right-sided facial tumour extending into the pterygoid fossa, forward along the superior alveolo-buccal sulcus and below to the submental region. The right tonsil was displaced to the mid-sagittal plane in the oro-pharynx. The mass measured 8·5 by 7·5 by 4·5 cm. No lymph nodal involvement was found. A biopsy had been taken from the most prominent portion of the tumour overlying the anterior margin of the mastoid process. The pathologist reported: "Anaplastic carcinoma of salivary gland". The tumour was obviously inoperable. Radiotherapy was given with the use of a supervoltage technique at 4,000,000 electron volts, an estimated minimum tumour dose of 5000 rads being delivered in four weeks. The boy is now well, with no clinical or X-ray evidence of malignant disease.

To achieve this result certain risks were involved. For example there may be retarded development of the skeletal structures of the right side of the face, jaw and maxilla. The secondary dentition may not erupt, or may consist of teeth which rapidly become carious. In fact, the patient may be demonstrated in twenty years, if he survives, as an example of undesirable sequelæ following radiotherapy.

Early medical workers using X rays and radium developed various degrees of skin damage, which often progressed to skin cancer. Luminous dial painters, who moistened their brushes on their lips, ingested small quantities of paint containing radium and mesothorium. These radioactive substances were largely deposited in skeletal structures, and this led to the development of bone tumours years later. Miners at Schneeberg in Saxony inhaled radium emanation and subsequently succumbed to lung cancer.

The immediate effect of radiation on the principal cellular elements of the blood is shown in Figure I. The number of lymphocytes in the peripheral circulation is diminshed within a few hours of a large exposure. The erythropoietic function is depressed much more slowly, and shows little, if any, change on routine radiotherapy applied to a limited volume of tissue. A long-continued occupational exposure to small amounts of radiation tends to produce a blood picture showing a relative granulopenia.

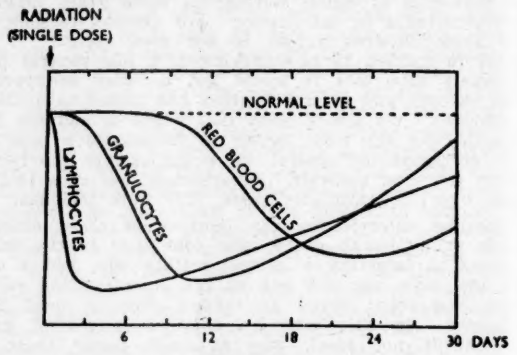

FIGURE I.

Effect of a single dose of X rays, about 300*r*, on the different cells in the blood.

In the industrial field, it is the practice to use potent sources of radioactive materials, mainly radiocobalt or radiocæsium, in small slugs for the radiography of metallic structures. It is inevitable that this procedure, unless confined to highly trained staff, will be accompanied by errors and misapplications. Reports of accidental human exposure in Australia have already been received.

It has been stated that 20% of the casualties following the atomic bomb explosions over Hiroshima and Nagasaki were due to gamma and neutron radiation, and that a single exposure of 500*r* of gamma radiation to the whole body constitutes a lethal dose to 50% of any population group exposed (Schubert and Lapp, 1957). The incidence of leukæmia in these two cities in the period up to 1954 was over three times greater than in the period prior to 1946 (Medical Research Council, 1956).

Fœtal Effects and Leukæmia.

It has been shown (Stewart *et alii*, 1956) that the incidence of cancer and leukæmia in children is higher after diagnostic X-ray examination or pelvimetry during the particular pregnancy. No survey of a similar nature has been published in this country. Prior to 1956 the history of ante-natal exposure to radiation was not elicited in cases of neoplasia in children, and it has been found that an attempt to obtain this retrospective information years after the demise of a child is disturbing to parents.

The following instances are briefly mentioned:

A female patient, aged 33 years, suffering from severe polycythæmia vera, was given radioactive phosphorus in

November, 1948, and the spleen, which was markedly enlarged, was treated by deep X-ray therapy. In August, 1949, the spleen which was then enlarged to the umbilical plane, was again treated by deep therapy with a single anterior field measuring 15 by 10 cm. and a total dosage of 750*r* on the skin in five applications of 150*r* over a two weeks period. At the time of this treatment the patient was at the end of her fourth month of pregnancy. When aged three years, the child developed acute megaloblastic leukæmia and survived for three months.

A child of six years was referred to me by Dr. Paul Hopkins, with lymphadenopathy which proved to be a reticulum cell sarcoma. It is known that the mother of the child had had a pre-confinement radiographic investigation.

The relationship between irradiation and the production of leukæmia will now be examined briefly. Sacks and Seeman (1947) reported that the leukæmia mortality showed an increase from 1900 to 1944, and the incidence of this disease in white patients has mounted in the United States of America from 42 per million *per annum* in 1940 to 68 per million in 1954 (U.S.A. Vital Statistics, 1956).

The four following groups of individuals provide evidence bearing on this matter: Group I: personnel exposed to neutron and gamma radiation at Hiroshima and Nagasaki; Group II: patients exposed to X-ray therapy for spondylitis; Group III: children exposed to X-ray therapy for thymic enlargement; Group IV: radiologists exposed to radiation, chiefly X and gamma rays.

Studies made by various researchers provide convincing evidence in each group that radiation is capable of inducing leukæmia.

Group I.

Table I indicates the incidence of leukæmia in the exposed populations of Hiroshima and Nagasaki. Attention is drawn to the lower incidence as exposure diminished with the increased distance from the hypocentre. The incidence for Zone D, where exposure was low, was 21 per million per year, which is the normal incidence in Japan.

TABLE I.

Annual Incidence of Leukæmia among the Combined Exposed Populations of Hiroshima and Nagasaki (January, 1948, to September, 1955) in Relation to Dose of Radiation (Gammas plus Neutrons); after Lewis (1957).

Zone.	Average Maximum Dose. (Rem.)	Incidence of Leukæmia per Million per Year.	Incidence of Radiation-Induced Leukæmia per Million per Year.	Probability of Leukæmia per Individual per Rem per Year.
A	1800	1200	1179	$0{\cdot}9\times10^{-6}$
B	500	390	369	$0{\cdot}7\times10^{-6}$
C	50	56	35	$0{\cdot}7\times10^{-6}$
D	5	21	—	—

Group II.

In the matter of leukæmia following the treatment by irradiation of spondylitis, the work of Court Brown and Doll (1957) is outstanding (see Table II). These workers estimated the expected incidence of leukæmia in a comparable group of unirradiated males to be 50 per million per year. Deducting this figure from the observed incidence in treated patients gives an estimate of the incidence of radiation-induced leukæmia.

Group III.

Group III is illustrated by the work of Simpson and others (Simpson *et alii*, 1955). These workers traced a series of 1400 infants who had received radiotherapy for thymic enlargement. The average lesion dose was estimated as lying between 100*r* and 300*r*. A control group of 1800 siblings of patients treated was traced. There were seven cases of leukæmia up to the age of 15 years in the irradiated subjects, and no case of leukæmia occurred in the control group.

Group IV.

The fourth group comprises approximately 3700 radiologists between the ages of 35 and 74 years, who practised in the United States of America. That a higher incidence of leukæmia existed in radiologists was first suspected and reported by March (March, 1944). The results of this survey are shown in Table III.

TABLE II.

Incidence of Leukæmia among Ankylosing Spondylitis Patients Receiving Different Doses of Radiation (X Rays); Data from Court Brown and Doll (1957); after Lewis (1957).

Maximum Dose to Spinal Marrow. (*r*.)	Estimated Average Maximum Dose. (*r*.)	Number of Males Developing Leukæmia.	Crude Incidence per Million Males per Year.	Incidence of Radiation-Induced Leukæmia per Million Males per Year.	Probability of Leukæmia per Individual per *r* (to Spinal Marrow) per Year.
0	—	—	50	—	—
Under 500 ..	—	2	220	170	—
500 to 999 ..	750	8	410	360	$0{\cdot}5\times10^{-6}$
1000 to 1499 ..	1250	8	420	370	$0{\cdot}3\times10^{-6}$
1500 to 1999 ..	1750	8	1130	1080	$0{\cdot}6\times10^{-6}$
2000 to 2749 ..	2375	6	1300	1250	$0{\cdot}5\times10^{-6}$
2750 or more ..	—	5	1760	—	—

Genetic Hazards.

The theory of genetic effects from radiation has been developed from a study of organisms and animals of short life, chiefly in fruit flies (*Drosophila*) and in mice. Some of these changes do not become manifest for several generations, and it is extremely difficult to provide clinical illustrations of genetic changes in humans who have undergone only two generations since Röntgen's discovery of X rays.

TABLE III.

Deaths and Death Rates from Leukæmia among Radiologists, at Ages 35 to 74 Years, by Five-Year Periods from 1938 to 1952, inclusive; after Lewis (1957).

Period.	Estimated Number of Radiologists at Mid-Point of Period.	Number of Deaths from Leukæmia.	Observed Death Rates per Million per Year.	Expected Death Rates per Million per Year.	Incidence of Radiation-Induced Deaths per Million per Year.
1938 to 1942	1451·5	5	690	101	589
1943 to 1947	(1850·7)[1]	6	650	(121)[1]	429
1948 to 1952	2250·0	6	530	141	389
(1938 to 1952)	(1850·7)[1]	17	610	(121)[1]	489

[1] The arithmetic average of the values for the 1938 to 1942 and 1948 to 1952 periods.

It is likely that other participants in this symposium will have dealt with the intriguing problem of "the doubling dose". Even if this could be estimated with any degree of accuracy, and if human populations were subjected to this exposure, it would take many generations for the frequency of genetic defects to be doubled. It has been stated, for example, that such a change in the genes would in one generation increase the frequency of low-grade mental deficiency by 3% and of schizophrenia and manic-depressive reaction by approximately 1%.

It is relevant to consider, however, that a radiation-produced gene will be recessive, and although it will be harmful in 99% of mutations, it will give no outward evidence of its existence until chance allies it with another allele of the same kind. This may not happen for many generations. The following instance of an exceptional case is quoted:

Mrs. A, aged 34 years, was first examined on January 25, 1944, the presenting history being that she had been treated medically for polycythæmia vera for two years. The patient

showed the classical features of polycythæmia vera with splenic enlargement of moderate degree. The blood count on her first attendance was as follows: the red blood cells numbered 8,900,000 per cubic millimetre, the hæmoglobin value was 170%, and leucocytes numbered 10,300 per cubic millimetre, 90% being neutrophils, 8% lymphocytes and 2% eosinophils. At that time whole-body irradiation was the mode of treatment advocated (Robbins, 1944). This article was reviewed in the "Year Book of Radiology" (1944) and the technique was commended by the editor in a special footnote.

The genetic dangers from irradiation were little understood, and the patient was given telerŏntgen therapy to the whole body. The factors used and the dosage administered were as follows: kilovoltage 190; filtration 3·0 millimetres of aluminium; focal skin distance 250 cm.; skin dosage anterior, 430*r*; skin dosage posterior, 250*r* (both skin dosages being administered in 22 days). The approximate ovarian exposure was 400*r* in 22 days.

A healthy infant was born in June, 1946, and the youth has been recently examined and found to be free of any clinically detectable defect and in excellent health. It is assumed that there are certain to be gene mutations of recessive type in both mother and child. The mother had a remission of four years after the X radiation, and has since been maintained in good health with radioactive phosphorus.

It is opportune to examine critically the practice of administering ovarian irradiation for sterility. The usual dosage to the ovaries is in the vicinity of 200*r*, and in some manner not understood about one-third of patients so treated subsequently bear children. On the assumption that a patient has three children, and if the "doubling dose" is taken as 40*r*, it can be calculated that these children on the average will carry one extra recessive mutation. The chance that the new mutant gene will be dominant is one in 100 (Schubert and Lapp, 1957). It is, therefore, unlikely that the first and second generations will be affected. Eventually, however, since "every mutation is a postponed disaster", one descendant will conceive a child who is homozygous for the new mutant gene, and a genetic death will result. A long series of minor disabilities may have occurred in the meantime. The situation has been summed up by Professor Muller, who gave the warning that "the frustration of present potential mothers would have been exchanged at the expense of an equal or greater amount of human frustration in later generations".

Safeguards for the Future.

Many of the untoward post-radiation effects mentioned in this short discourse followed treatment applied before the practice of radiotherapy was accredited as a special field of medicine. It must be recalled that the outstanding work of Paterson and Parker on radiation dosage was published in 1934. Prior to this, the application of radium was carried out by arbitrary methods and generally by untrained practitioners.

Staff exposed to X or gamma radiation now wear film badges. The maximum permissible body exposure is taken as 0·3*r* in a week; but such exposures cannot be permitted to continue at this rate. In practice it is common to see most staff registering under 0·05*r* per month when working in a department where full attention is paid to protective measures.

It is impracticable to devise adequate lead protection against gamma rays in operative procedures, and prior to 1954 a radiotherapist carrying out a large implant with radium needles or radon seeds was exposed to 0·1 to 0·3*r* in the course of carrying out one implantation. Moreover, in the performance of a cylindrical implant of the rectum or vagina with the use of the left forefinger as a guide, this digit was exposed to 5·0 or 10·0*r* in a twenty-minute period. This can now be obviated by the after-loading technique described in Australia by Mowatt and Stevens (1956). Hollow needles are used, and are quickly charged after implantation by the insertion of the pre-calculated radon sources.

Radiotherapists are striving persistently to eliminate or to diminsh hazards, as the following example serves to illustrate. Until 1946 the treatment of epithelioma of the ocular conjunctiva was the removal of the affected eye. Treatment by excision of the lesion and application of a radon ring was followed by a high percentage of cures; but the development of lens opacities was a frequent complication. Since 1952 this condition, the incidence of which is relatively high in Queensland, has been very successfully treated by a radioactive strontium beta-ray source held in a special applicator (Mead and Robertson, 1957).

TABLE IV.

Summary of the Genetically Effective Average Gonad Doses from Medical Diagnostic X-ray Examinations and Radiation Therapy Treatments Received per Person during One Generation (30 Years) by the Population of the United Sattes of America.[1]

Source of Radiation.	Minimum Gonad Dose. (*r*.)	Probable Gonad Dose. (*r*.)
X-ray diagnostic examinations:		
Radiography	1·0	1·8
Fluoroscopy	0·3	1·5
Photofluorography and mass chest X-ray examinations	0·006	0·006
Dental X-ray examinations	0·03	0·1
Obstetrical X-ray examinations	0·16	0·7
Total diagnostic dose	1·5±1	4·1±3
X-ray and radioisotope therapy treatments	0·5	0·5
Total gonad dose	2·0	4·6

[1] The minimum average doses have been computed on the basis of the lowest gonadal doses reported. Even further reduction can be obtained, since improved techniques are used for some procedures for which the gonad doses are not yet measured. The probable average doses are based on an average of those reported measurements of techniques generally employed.

A plea is made for a pooling of knowledge gained from research efforts in the Western world, as there are still many gaps in our understanding of the biological effects of radiation. Full attention should be given to the recommendations of the International Commission on Radiological Protection. Occupational limits of safe exposure should be reviewed downwards in the direction of 0·1*r* per week.

TABLE V.

Summary of the Average Radiation Doses to the Gonads Received in 30 Years by Persons Living in the United States of America; after Schubert and Lapp (1957).

Radiation Source.	Average 30 Year Gonad Dose.[1] (*r*.)
Natural background	5·0 (2·4 –6·2)
Fallout	0·5 (0·02–0·4)
Medical examina?ions	10·0 (1·0 –5·0)
Miscellaneous	0·3 (0·0 –0·03)

[1] Values in parentheses represent the range of values listed by the Genetics Committee.

Every effort should be made to reduce exposure to the general population. In England there are 60 pelvimetries and 200 pre-confinement X-ray examinations per 1000 births. It is difficult to justify a continuance of this practice.

An estimate of the average radiation exposure to the gonads received in a period of 30 years by persons living in the United States of America is 16 *r* (Schubert and Lapp, 1957). Of this total, an estimated 5 *r* are derived from natural background and 10 *r* result from medical examinations (Table IV).

It is held that a measure of control should be applied to the use of therapeutic and diagnostic X-ray apparatus, especially in the hands of persons who have no radiotherapeutic or radiological training. The use of shoe-fitting X-ray machines should be banned.

Parliamentary enactments are being implemented in most countries to control the usage of radioactive substances and isotopes. These are praiseworthy develop-

ments; but few of these measures reach out into the field of diagnostic röntgenology, from which the general population receives most of its radiation. Is it not opportune for the medical profession to draw up its own code of radiation practice, before an informed association of patients undertakes to recommend methods of management to medical practitioners?

Summary.

Certain late radiation effects have been reviewed and the genetic hazard has been discussed. Some possible safeguards for the future have been mentioned.

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SOCIAL MEDICINE RESEARCH METHODS.[1]

By ALICE STEWART,
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SOCIAL medicine research (which covers many aspects of epidemiology, human genetics and physical anthropology) must be based on human data and is, therefore, compelled to take the form, not of planned experiments, but of surveys. The main use of these surveys is to demonstrate the existence of genetic or environmental influences which are capable of interfering with normal development or altering the prevalence (but not the basic pathology) of diseases, and to measure their effects in these respects.

In order to prove that something (e.g., the tubercle bacillus) is responsible for a particular set of pathological changes (i.e., is acting as a specific cause of a disease), it is necessary to apply tests of biological specificity — for example, Koch's postulates. If, however, a factor merely alters the prevalence of a disease, such tests are inappropriate. In this context, the experience of animals is never completely relevant, and the endpoints of the suitable investigations can be only the demonstration of statistical relationships between presumed risk factors (which may be genetic or environmental in origin) and presumed medical consequences (which may be a disease or the outcome of a medically important situation, such as childbirth, childhood or puberty). If applied to a disease of unknown ætiology, this method may indicate the specific cause; but proof of its existence can never be established within the framework of the original investigation.

Surveys are not unique to social medicine, nor are they confined to studies of disease causation. The same methods can be adapted to study such things as the effects of disease, the effects of treatments or preventive measures and the effects of medical services.

Surveys follow the set pattern of (i) formulating a working hypothesis based on one or more arbitrary assumptions, (ii) testing these assumptions by collecting uniform data from test and control groups, and (iii) examining these facts for evidence of statistically significant relationships.

Test and control groups may be defined in terms of the risk factor or in terms of the medical consequence; but because one is dealing with human situations, it is never possible to get "clean" groups. The only feasible proposition is to examine situations which are relatively alike in respect of the people who represent them, and relatively dissimilar in respect of the risk factor. There is always a risk that unknown bias may affect the results of a survey, and this must be tested for in various ways. For instance, a retrospective survey may be followed by a prospective one, in which the working hypothesis of the findings of the former are incorporated.[1] But perhaps the best test of reliability is for a number of independent observers to examine the same problem, using different groups and different methods.

If repeated surveys show that one risk factor only alters the prevalence of one disease, the former is bound to be closely related to the specific cause of the latter. However, most naturally occurring conditions have non-specific medical effects. It follows that, in general, social medicine research is directed towards the quantitative aspects of disease, and pathological and clinical research to the qualitative aspects.

Collection and Coding of Data.

The facts required in any survey fall into two groups — those which are needed to prove an association between the risk factor and the medical consequence (contrast data), and those which are needed to reveal unwanted differences between test and control groups (standardizing data). Since the latter must be treated as presumed risk factors until proved otherwise, it follows that all the data must be treated in the same way. If this is done, the final statistical analysis will reveal the extent to which accidental differences have masked or exaggerated the effects of the designated risk factor.

The facts required may be collected *de novo* (raw data), or they may be derived from statistical digests of facts collected by someone else before the investigation began (processed data). It is not necessary (though it may be convenient) for test and control groups to contain equal numbers of people, nor for them to remain the same throughout the investigation. One situation may provide good opportunities to study one facet of a problem but prove inadequate in other ways, and there is no reason why a survey should not consist of several different approaches to the same problem and incorporate both retrospective and prospective data. The best known sources of processed data are official statistics of mor-

[1] Read at the North Queensland Medical Conference, Mackay, September 22 to 29, 1958.

[1] Definitions: a retrospective survey is one in which the presumed medical consequence predates the collection of data; a prospective survey is one in which it post-dates the collection of data.

tality. These find their greatest use in the planning stage of a survey, but they can also form part of the data needed to compare test and control groups.

Interviews are not an essential part of a survey, but they play an important role, and a knowledge of the principles underlying the design of standardized schedules or *questionnaires* is useful. The basic characteristic of these documents is an arrangement whereby all the facts required have an equal opportunity of being accurately recorded. As these facts have eventually to be translated into numbers, it is often convenient to offer a choice of answers and to ask the interviewer to ring a number. But experience suggests that the more ingenious a form is in this respect, the more useless it becomes. All but the simplest facts should be recorded in full at the time of collection, and only later translated into code numbers. The latter fall into two categories: the purely descriptive numbers (e.g., sex, occupation, diagnosis), and numbers representing a scale of values (weight, hæmoglobin value, skeletal maturity, intelligence quotient). The scale of values may already exist, but an investigator must always be prepared to invent his own yardsticks.

In the final stages of a survey, all the facts must be available in the form of punch cards which can be sorted on electrically operated machines; but during the intermediate stages it is often advisable to arrange them in such a way that the records for different individuals appear in vertical sequence and the different items of information in horizontal sequence (ledgering system). In this form not only are the facts easier to check, but they are arranged in such a way that it is a comparatively simple matter to work out means, standard deviations and coefficients of correlation, or to condense all the facts into a set of standard deviation units. Ledgering is a particularly useful device when, as in growth studies, the medical consequences have to be measured over a series of years, for in this form it is a comparatively simple matter to turn static measures (e.g., weight at a given age) into dynamic measures (e.g., weight increments for given periods). Such measures are an essential part of all dynamic or longitudinal surveys (i.e., surveys in which serial observations are made on the same groups of people).

For obvious reasons, the ledgering system is not applicable to large-scale surveys; but it does serve a useful purpose if the numbers of people are, say, less than 2000.

From what has been said, it should be obvious that the final stages of a survey consist in going meticulously through the records in search of cross-correlations and systematically relating these to the laws of statistical probability. Finally, in the light of what is discovered, conclusions must be drawn which not only accord with these laws, but make medical sense. It will be a mark of success if these conclusions stand the test of time and prove to be useful sign-posts for workers in other, more fundamental, branches of research.

Other Uses of the Term Social Medicine.

In the foregoing account, the term social medicine has been used to describe a technique which has only recently attracted much attention, but which holds considerable promise as a means of studying the medical effects of genetic and environmental influences. But the words are also used to describe recent changes in medical "welfare" practice, in public health or the "new look" in clinical medicine as practised in hospitals. To appreciate why it has been thought necessary to coin a new term to describe two old-established services, it is important to realize that the medical needs of post-war society in England are very different from the needs of other times and other societies, and that, in consequence, there has been a minor revolution within the medical profession. As with other revolutions, this one has thrown into sharp relief doctors who welcome the new régime and feel the need for new slogans, and doctors who resent the changes and insist that the slogans merely describe what was being done satisfactorily in the past.

The relatively abrupt changes which have characterized recent years are partly due to circumstances outside the control of the profession — namely, the high standard of living which England now enjoys, and the redistribution of wealth and education which has occurred in recent decades. But they are mainly the result of its being possible, for the first time in history, to cure a large number of potentially fatal diseases and to institute on a wide scale effective measures for disease control. This has enormously accelerated the change-over from what may be called a purely curative approach to medical problems to a curative-cum-welfare approach. The most obvious expression of the change is the establishment of the National Health Service in 1948; but in fact it began in the nineteenth century and is still progressing.

There have been several attempts to describe the adjustments that the medical profession is making to keep abreast of society, and these are reflected in the current fashion of insisting that "medicine is part of society" (self-evident fact), and of using the term "social medicine" to describe the work of doctors who are most directly concerned with the welfare aspects of contemporary medical practice. This fashion has certainly caused some confusion to the present generation of doctors; but it is reasonable to forecast that future generations will have no use for our slogans, and will be content to use the term "social medicine" to describe a method of research which has in the meantime become better known and more appreciated.

NEW THOUGHTS ON OBSTETRICAL HÆMORRHAGE.[1]

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Much of what I have to say will deal with the diagnosis and treatment of blood coagulation defects as encountered by the obstetrician, and typical case records will be quoted; but I should like to refer briefly to the diagnosis and management of placenta prævia, to the conduct of the third stage of labour, and to the problem of achieving greater fœtal "salvage" in cases of accidental hæmorrhage.

Placenta Prævia.

The superiority of the expectant treatment of placenta prævia is now well established, and the need to refrain from premature vaginal examination to confirm the diagnosis is generally accepted. When the amount of bleeding calls for active intervention, the choice of procedure is now virtually limited to Cæsarean section in the more severe degrees of low placental implantation and artificial rupture of the membranes in the remainder. Some obstetricians prefer to restrict the latter procedure to cases in which labour has already begun, and in which an easy, quick delivery can be anticipated.

Constant efforts are being made to improve the methods of diagnosing placenta prævia. Although refinements in X-ray diagnosis are being developed, the importance of certain clinical features is being increasingly stressed, particularly the significance in the last trimester of recurrent malpresentation and of an unaccountably high head, whether or not the typical painless self-arresting ante-partum hæmorrhage has occurred. Soft-tissue placentography calls for highly specialized techniques and is probably unsuitable for general application. Of more universal application are the techniques used to study on a lateral film anterior or posterior displacement of the fœtal head as a result of a low-

[1] Read at the North Queensland Medical Conference, Mackay, September 22 to 29, 1958.

lying placenta. Needless to say, a reasonable degree of maturity is necessary before this sign is significant. Cystography has been abandoned. The latest trend towards localization of the placenta is by the use of radioactive sodium, the count being appreciably higher over the placental area, but the method has as yet no clinical application.

When the patient has been under expectant treatment, an increasing number of obstetricians now believe that they can arrive at a correct diagnosis without having to introduce a finger through the cervix, by taking into consideration the history, the position of the presenting part and the X-ray findings. Nevertheless, there is a group of cases in which the diagnosis still remains in doubt unless one feels for the edge of the placenta with a finger through the cervix, under appropriate circumstances. By exercising great gentleness the obstetrician can reduce the amount of blood loss, especially if, by using fundal pressure, the placental edge is brought down on to the finger instead of the finger's reaching upward for the placenta.

Accidental Ante-Partum Hæmorrhage.

It is, however, in connexion with accidental ante-partum hæmorrhage that methods of treatment have undergone significant changes, and this is in part due to recently acquired knowledge of coagulation defects occurring under these circumstances. There is a good deal of discussion on the most useful place for Cæsarean section in accidental hæmorrhage as a means of reducing fœtal loss. In the presence of long-standing hypertensive vascular disease, retroplacental hæmorrhage is apt to occur early in the last trimester, and before fœtal viability is really established. Very little further "fœtal salvage" can be effected in this category. At the other end of the scale are those patients whose accidental hæmorrhage occurs for no very obvious reason, or perhaps in association with preeclamptic toxæmia; such an episode is more likely to occur during the last six weeks or so of pregnancy. These patients often come into labour spontaneously after the hæmorrhage, or can be readily brought into labour by artificial rupture of the membranes, and, provided that intrapartum fœtal anoxia can be prevented by appropriate blood transfusion, and by the intranasal administration of oxygen to the mother, together with the intravenous administration of fibrinogen when indicated, fœtal loss need not be high.

It is appreciated, however, that there is a comparatively small residue of cases in which the accidental hæmorrhage occurs after fœtal viability has been attained, in which the fœtal heart sounds, though disturbed, are still present, but in which the cervix remains firmly closed. Spontaneous labour does not ensue, and experience suggests that in the absence of intervention the fœtus will surely die.

There are, of course, complicating factors, such as the presence of some degree of placental insufficiency and a poorly developed fœtus in spite of the period of gestation. However, with the possible exclusion of the latter, it is from this small residue of cases that, in the opinion of many obstetricians, timely Cæsarean section will ensure a live infant. Needless to say, the decision to effect delivery in this way should not be deferred for very many hours, because of the risks of neuronal damage to a fœtus exposed for many hours to a non-lethal but highly damaging degree of hypoxia. Any demonstrable coagulation defect must, of course, be corrected before surgical delivery is undertaken.

I would add, in connexion with accidental ante-partum hæmorrhage, only that the grave sequelæ sometimes encountered in the past — i.e., anterior pituitary necrosis, the Couvelaire uterus and anuria due to symmetrical cortical necrosis of the kidneys—will, it is anticipated, be encountered less commonly in the future, because of the early recognition of coagulation defects, the adequate treatment thereof, and the consequent limitation on the rate and amount of maternal blood loss and on the extent of utero-placental damage.

Post-Partum Hæmorrhage.

There have been significant changes, too, in our appreciation of the physiology and management of the third stage of labour and in the management of established post-partum hæmorrhage. It is now believed that the placenta begins to separate almost at once, and that separation is complete within four to six minutes of the birth of the baby.

Oxytocic drugs, particularly ergometrine, are frequently given before the delivery of the placenta; Credé's expression of the placenta is seldom performed; manual removal is carried out earlier and more readily and even prophylactically, and blood transfusion is used more freely. Occasionally, when this is indicated, coagulation defects are being treated by the intravenous administration of fibrinogen. This is not the time to evaluate in detail these current trends, but I should like to make one or two points.

There are a number of obstetricians who believe that prophylactic manual removal should be carried out on any patient who has been fully anæsthetized for a low mid-pelvic forceps delivery, breech extraction or other complicated delivery. They believe that the chances of uterine atony, retained placenta and post-partum hæmorrhage are very real in such cases, and that the risks of a second anæsthetic cannot be minimized, and they therefore give ergometrine (0·5 mg. by intravenous injection) and remove the placenta under the same anæsthetic. Prophylactic manual removal may also have a place in the management of patients with a history of habitually retained placenta with post-partum hæmorrhage.

In spite of the wisdom of avoiding unnecessary delay, it is hoped that the current trend towards early (but not prophylactic) manual removal of the placenta will not be carried to extremes, for should the need for manual removal develop rapidly by reason of excessive blood loss immediately after delivery, the practitioner would nevertheless do well to stay his hand until the condition of the patient has improved. Ergometrine given by the intravenous route will control the rate and amount of loss, and the patient's blood pressure must be restored by suitable intravenous therapy before manual removal is begun.

Much has been written for and against the routine use of oxytocic drugs prior to the completion of the third stage of labour. I will not attempt to review this evidence; but I would say without hesitation that the selective rather than the routine use of this measure seems to achieve the best results. I should give ergometrine intravenously immediately after the birth of the child or within the next few minutes to any patient who already had begun to show evidence of post-partum hæmorrhage to the extent of five fluid ounces or more of blood loss, to any patient with a history of previous post-partum hæmorrhage, and in any case in which recent ante-partum hæmorrhage had occurred. I consider it a useful alternative to prophylactic manual removal after delivery requiring full surgical anæsthesia, and I consider it mandatory in the presence of pregnancy anæmia, excessive parity, poor general physique, multiple pregnancy, and previous hydramnion.

Obstetrical Hæmorrhage Associated with Defibrination.

Perhaps the most significant change in the management of obstetrical hæmorrhage has come about as the result of our fuller appreciation of blood coagulation defects which occur under certain circumstances, and the discovery of a satisfactory method of combating this state of affairs.

Afibrinogenæmia (or more commonly, hypofibrinogenæmia) is a condition in which the blood is rendered incoagulable owing to the absence or gross deficiency of one of the principal components of the normal blood-clotting mechanism, with a resultant hæmorrhagic diathesis. It may be seen in at least three obstetrical circumstances, and I shall refer briefly to typical case histories in each category. These include amniotic

fluid infusion, severe premature separation of the placenta, and long-standing retention of a dead fœtus in utero. The significant hæmatological change which occurs in these cases is defibrination of the blood, and the exciting factor postulated is the substantial access of thromboplastin into the maternal circulation, with the subsequent occurrence of intravascular blood coagulation and consequent defibrination. In some instances excessive fibrinolytic activity contributes further to the defibrination.

The normal fibrinogen content of plasma appears to vary from 200 to 250 mg. per 100 ml.; but this level is raised in pregnancy, and the mean normal fibrinogen level at term is about 300 mg. per 100 ml. The minimum level for effective hæmostasis is considered to be in the region of 100 mg. per 100 ml. of plasma. The average total fibrinogen content of plasma in a normal healthy person is thought to be between eight and 12 grammes.

Although a great deal of new work has been done on the subject of blood coagulation, it will be assumed for the purpose of this discussion that the essential reaction in coagulation of the blood is the conversion of the soluble protein fibrinogen into the insoluble protein fibrin by means of the enzyme thrombin. For the activation of prothrombin to thrombin, free calcium ions and certain organic activators of obscure composition named thromboplastins are necessary. These thromboplastins are derived from damaged tissues, and the damaged uterus and the damaged placenta are particularly rich in thromboplastic material. The latter is also found in amniotic fluid, possibly from fœtal exfoliation.

Defibrination Associated with Amniotic Fluid Infusion.

It is now realized that patients with amniotic fluid infusion (previously termed amniotic embolism) may die from hæmorrhage as well as from respiratory distress; but before this syndrome is considered in detail, it would be useful to trace briefly the development of our understanding of obstetrical hæmorrhage associated with defibrination. That coagulation defects could be a cause of apparently uncontrollable bleeding in obstetrical practice was first recognized by De Lee in 1901, who termed the condition "temporary hæmophilia"; but it was not until 1936 that Dieckmann, also of Chicago, clarified the position by demonstrating that a state of hypofibrinogenæmia existed in a number of patients suffering from concealed accidental hæmorrhage, especially those in whom the bleeding continued despite all recognized methods of treatment.

The next development was along slightly different lines—soon, however, to show points in common with the syndrome described by Dieckmann. The year was 1941, and from the United States (Steiner) and subsequently from the United Kingdom came reports of maternal pulmonary embolism by constituents in amniotic fluid as a cause of "obstetric shock" and of sudden unexpected maternal deaths during and shortly after parturition.

The essential autopsy finding in these cases was widespread embolism, involving the smaller pulmonary arterioles from meconium, globules of vernix caseosa and other particulate matter normally present only in amniotic fluid.

At the time this was regarded as a new obstetric syndrome, and it was thought that these cases had been formerly regarded as deaths from "obstetric shock" or "idiopathic" post-partum hæmorrhage or acute pulmonary œdema. Further cases of so-called "amniotic embolism" were reported in the literature, especially between 1948 and 1950, and those patients who survived the initial "embolic" episode were noted to have a tendency to uterine bleeding which was not readily controlled.

Since 1950 there has been a growing belief (Torpin, Roby, Reid, Weiner) that the autopsy findings of particulate matter, normally present only in amniotic fluid, within the pulmonary arterioles, is only incidental to a more widespread disturbance, and that the deaths reported were not in fact "embolic deaths" in the usual sense of the word. The condition thus came to be known as amniotic fluid infusion, with the emphasis on the clinical consequences of massive infusion of amniotic fluid into the maternal circulation. It is suggested that the fluid finds its way into the maternal blood-stream, either through a tear in the membranes near the edge of the placenta, or through torn endocervical veins. In either case, however, it is postulated that the dilating cervical canal is firmly plugged by a large and well-fitted fœtal head, that strong uterine contractions are present, and that for the most part some marginal separation of the placenta has occurred.

The consequences of amniotic fluid infusion are either sudden death associated with parturition, or, if recovery occurs from the initial "shock", hæmorrhagic manifestations, usually in the form of a persistent and apparently uncontrollable post-partum hæmorrhage. Evidence is accumulating to suggest that those who die within a few minutes do so because of extensive intravascular coagulation of blood, and that those who die later from hæmorrhage, or who recover after partial exsanguination, bleed uncontrollably because they are subject to a gross defibrination, which brings their blood level of this constituent into the hæmorrhagic range.

The process is regarded as being initiated by the thromboplastic activity latent in amniotic fluid, should it gain material access to the maternal circulation. The extent of the intravascular clotting and subsequent defibrination will determine the clinical picture, which is one of profound shock occurring suddenly and unexpectedly in a woman, usually a multipara with a large infant, who is, or who has recently been, in unusually strong labour. There is some suggestion of an association, by no means constant, of this syndrome with the medicinal induction of labour. If the initial "shock" is rapidly followed by dyspnœa, marked cyanosis, vomiting and restlessness, the clinical picture is more complete. The rapid onset of signs of pulmonary œdema in the known absence of pregnancy toxæmia, cardio-vascular or renal disease would further support the diagnosis. In those cases in which the patient survives the initial episode, recurrence or continuation of post-partum uterine bleeding gives further support to the diagnosis of amniotic fluid infusion, especially if such loss cannot be controlled by the intravenous administration of oxytocics, together with intrauterine packing and massive blood transfusion.

The treatment of this condition is chiefly concerned with the control of the hæmorrhagic state which develops in those patients who survive. In the presence of presumptive clinical evidence of this syndrome, and on the assumption that the initial respiratory distress and shock have received appropriate symptomatic treatment, and that hæmorrhage is now the problem, it is essential to take the following steps:

1. To effect an early restoration of blood volume. Stored citrated blood is usually the most readily available agent for this purpose (and should most certainly be used); but clinical experience has shown that transfusion alone, even in large amounts, is insufficient to change the clinical picture. Synthetic plasmas, such as "dextran" or "plasmosan", are best avoided in any quantity, as they may aggravate the condition. However, the infusion of one half-litre bottle only is unlikely to do this, and will allow time for cross-matching of blood.

2. To test the clotting time and clot retraction behaviour of the patient's blood. This simply means incubating for half an hour to one hour, at 37° C., 5 ml. of the patient's venous blood in a glass test tube. In the presence of afibrinogenæmia there will be no clot formation, but the addition of fibrinogen will cause the blood to clot. In the presence of hypofibrinogenæmia, clot formation will be minimal and fragmentary, or dissolution of the clot may occur within an hour after the blood is withdrawn.

The technique is obviously a very simple one, and laboratory facilities are not required, as the observations

can be made if necessary at room temperature by immersing the test tube in water maintained at 37° C.

A simple laboratory test has been advised by Bonsnes and Sweeney (1955). In simple terms, a recently prepared amount of thrombin is added to a sample of the patient's plasma. In the presence of normal fibrinogen levels, clotting will occur almost immediately—i.e., within 60 seconds. In the absence of fibrinogen, clotting will not occur, and in its relative absence clotting will be delayed. A control test is performed at the same time.

3. To administer human fibrinogen by the intravenous route. The way is now clear to do this with the diagnosis confirmed beyond doubt. It is necessary to emphasize that it would be unwise to attempt to make a firm diagnosis of defibrination simply from the fact that the blood being lost per vaginam was not clotting. The maximum amount which may theoretically be given is that equivalent to the total fibrinogen content of circulating blood (eight to 12 grammes), as complete defibrination is possible. However, it is usual to commence with four to six grammes, subsequent dosage being determined by the response obtained. The administration of one gramme at half-hourly intervals until the blood-clotting mechanism has returned to normal has been suggested.

The following two case histories (which are presented in a summarized form) are typical of this syndrome. Case I was undoubtedly one of massive amniotic fluid infusion with rapid deterioration and death, and Case II illustrates amniotic fluid infusion with recovery, followed by persistent uterine hæmorrhage associated with demonstrable defibrination, and recovery after the intravenous administration of human fibrinogen.

CASE I.—Mrs. A., aged 40 years, was suffering from chronic hypertensive vascular disease; she was pregnant for the seventh time. She was admitted to the Brisbane Women's Hospital after 36 weeks' gestation, the blood pressure reading at that time being 180/90 mm. of mercury. By the thirty-eighth week this figure had risen to 170/105 mm. of mercury, and, as the condition of the cervix was suitable, the forewaters were ruptured; the liquor was stained with meconium. Because labour had not commenced 72 hours later, a single injection of 3 min. of oxytocin was given. Violent contractions developed, and within an hour the patient was delivered precipitately of an infant weighing 9 lb. The third stage of labour was normal; but ten minutes later the patient was restless and cyanosed, and free bleeding per vaginam had commenced. This was accompanied by a marked fall in blood pressure to 60/40 mm. of mercury and marked respiratory embarrassment. The fundus uteri remained firm. In spite of adequate symptomatic and restorative treatment the patient's condition rapidly became worse, and death occurred one and a quarter hours after the initial collapse.

At autopsy there was histological evidence of epithelial cells and fat globules in the pulmonary arterioles.

CASE II.—Mrs. B. was a primipara, aged 34 years, whose pregnancy had been uneventful, and for whom medicinal induction of labour had been prescribed after her admission to the intermediate section of the Brisbane Women's Hospital, the indication being post-maturity. Strong uterine contractions had developed almost immediately after the first injection of oxytocin, and this tendency continued throughout the subsequent labour, which was completed within eight hours, the infant weighing 8 lb. 2 oz. A moderately severe post-partum hæmorrhage occurred; but this was adequately controlled by the intravenous administration of ergometrine.

A little less than an hour later the patient suddenly collapsed, with marked cyanosis of the lips and extremities, gross respiratory embarrassment, an impalpable radial pulse, and crepitant râles heard at both lung bases. There was no further blood loss at this stage. Symptomatic and restorative treatment over the next two hours led to a considerable improvement in the patient's condition; but, as the blood pressure rose to a recordable figure, vaginal bleeding recommenced. During this period 5 ml. of venous blood had been incubated at 37° C., and on periodic inspection of the sample there was no indication of any clot formation whatever. Six grammes of human fibrinogen were therefore given intravenously, followed by a further 1·5 litres of compatible blood. However, the blood loss per vaginam continued unabated, and a second synchronous transfusion was commenced.

Four hours after the initial collapse the blood pressure was recordable at 60/30 mm. of mercury, and towards the end of this period a fresh sample of venous blood clotted within six minutes, and the blood being lost per vaginam showed a greater tendency to clot. Ten hours after the initial episode a blood pressure reading of 100/60 mm. of mercury was obtained, and no further significant vaginal blood loss occurred. Occasional râles were still audible at the base of the left lung.

For the next 48 hours the patient was pyrexial, and complained of marked blurring of vision. This resolved completely, and further progress was towards complete recovery.

Defibrination Associated with Accidental Hæmorrhage.

Those patients who die from concealed accidental hæmorrhage usually do so from shock, and from the immediate and extensive blood loss associated with the more severe manifestations of this disorder. The remaining deaths usually follow renal or pituitary damage, either of which is initiated by the vascular accident. With the prognosis so closely bound up with the extent of the initial shock and the ability or otherwise of the medical attendant to control the hæmorrhagic manifestations, and because impaired blood coagulability had been noted for many years with the more severe forms of this condition, it is not surprising that the concept should be put forward that defibrination should sometimes enter into the picture of premature placental separation with persistent and apparently uncontrollable bleeding, whether before or after the expulsion of the fœtus.

The fundamental disturbance is again believed to be fibrinogen depletion, and this in turn is presumably initiated by the introduction of thromboplastic material, derived in this instance from the damaged uterus and placenta, into the systemic circulation. The basal decidual plate is, of course, ruptured under these circumstances.

It is hardly necessary to add that any alteration in the clotting mechanism is believed to follow rather than to precede or initiate the retroplacental hæmorrhage. Once the patient has been safely delivered, the introduction of thromboplastic material into the circulation is relatively abated, and the extent of the coagulation defect does not proceed further in spite of the damage sustained by the myometrium. However, it may have proceeded sufficiently far prior to delivery to cause fatal post-partum hæmorrhage in the absence of effective treatment.

The conception that defibrination is often present in the more severe types of accidental hæmorrhage, especially in the condition previously known as the Couvelaire uterus, has completely altered previous ideas about the place of Cæsarean hysterectomy in such cases. Moreover, unless the clot observation or Bonsnes test has shown normal coagulation, the advisability of early Cæsarean section in the interests of the infant is also contested. It is now suggested that the possibility of defibrination be considered in these cases of severe accidental hæmorrhage in which the patient, either still unconfined or having been delivered, is not responding to the usual restorative measures, and in which evidence of continuing blood loss (even though wholly concealed) is present.

Spontaneous bleeding from the gums or bowel or tissue bruising would support a diagnosis of defibrination. Such a clinical picture would naturally lead to the setting up of a clot observation test, with or without the addition of thrombin. It is now standard practice at the Brisbane Women's Hospital to carry out the former test in all cases in which a diagnosis of accidental ante-partum hæmorrhage has been made.

Because the uterus often becomes hypertonic for many hours after an accidental hæmorrhage, and because it is envisaged that this hypertonus will facilitate the further access of thromboplastic material into the maternal blood stream, artificial rupture of the membranes is believed

to play an important part in treatment by reducing intrauterine tension and promoting the onset of labour.

The following two case histories are typical of the syndrome of defibrination associated with accidental hæmorrhage. Case III is of interest because the plasma fibrinogen level was subsequently shown to be as low as 50 mg. per 100 ml. This case also serves to illustrate the fact that the establishment of apparently normal clotting power after the administration of 4 grammes of fibrinogen prior to delivery seemed to be insufficient to prevent an alarming post-partum hæmorrhage some 24 hours later. It is probable that fibrinolysins had developed during the intervening period, and had thus nullified to some extent the improvement brought about by the initial 4 grammes of fibrinogen.

CASE III.—Mrs. C., aged 29 years, was pregnant for the third time, her previous pregnancies having been complicated by mild preeclamptic toxæmia on each occasion. She was admitted to the Brisbane Women's Hospital after 35 weeks' gestation in a condition of shock. Her blood pressure was 85/60 mm. of mercury. The uterus was distended and tender, and a diagnosis of concealed accidental hæmorrhage was made. Five millilitres of venous blood taken shortly after admission had not clotted after one hour at 37° C., and the result of the Bonsnes test was positive. Four grammes of fibrinogen were given, and a subsequent clot observation test showed good clotting after eight minutes. In the meantime blood transfusion had been continued, and the patient's blood pressure had risen to 120/80 mm. of mercury.

Labour pains commenced the following day, and a stillborn fœtus was delivered some 30 hours after the patient's admission to hospital. The delivery was followed by a rather alarming post-partum hæmorrhage necessitating recommencement of the transfusion therapy. Subsequent progress was uneventful. It was later reported that on the day of her admission to hospital the patient's plasma fibrinogen level had been 50 mg. per 100 ml.

Case IV is typical of our experience with this condition, and it may be significant that a total of 8 grammes of fibrinogen were needed.

CASE IV.—Mrs. D., aged 32 years, was pregnant for the second time, her first pregnancy having been complicated by a small ante-partum hæmorrhage. She was admitted to Brisbane Women's Hospital after 32 weeks' gestation as an unbooked patient, with the typical manifestation of a moderately severe accidental hæmorrhage. The fœtal heart sounds were not present. The patient's blood pressure was 130/90 mm. of mercury. During the next few hours there was evidence that the retroplacental hæmorrhage was increasing in amount. The bleeding also became revealed. Venous blood incubated at 37° C. showed minimal and fragmentary clot formation after 30 minutes. Four grammes of fibrinogen in 500 ml. of normal saline were given, but this measure produced little change in the clinical picture. The uterus was extremely tense, and there was continuous blood loss per vaginam.

There was no sign of commencing labour. The patient's general condition was far from satisfactory and signs of progressive shock were now present. Another 4 grammes of fibrinogen were added to the blood transfusion which was being given; a subsequent clot observation test showed firm clotting after five minutes, and this sample remained clotted after one hour.

In the meantime there had been a gradual but steady improvement in the patient's condition, and this was accompanied by signs of commencing labour. The membranes ruptured spontaneously, and a stillborn infant was delivered some 24 hours after her admission to hospital. There was no significant post-partum hæmorrhage, and subsequent progress was uneventful. A total of 8 grammes of fibrinogen and 2·5 litres of citrated blood was given during the immediate ante-partum period.

Defibrination Associated with Long-Standing Intrauterine Fœtal Death.

There have now been a number of well-authenticated case reports, in which patients who have retained a dead fœtus in utero for some weeks have bled alarmingly during delivery or in the immediate post-partum period. The amount of placental autolysis following fœtal death in utero varies greatly, but it is seldom absent, and, as we have seen, the placenta contains considerable quantities of thromboplastic material.

This autolysis sooner or later in some instances involves the basal decidual plate. It is postulated that fragments of disintegrating, but actively thromboplastic, placenta thus enter the maternal circulation intermittently under pressure of the painless contractions normally present during the waiting period, or as the result of the actively contracting uterus when labour supervenes. It is probable that under these circumstances, in which access of thromboplastin to the maternal circulation is spread over a considerable period of time, the intravenous clotting takes the form of a thin layer of fibrin deposited over the intima of the vascular system.

From the clinical point of view it is apparent that, whilst defibrination is a relatively rare complication of intrauterine fœtal death, it is probably wise to estimate the fibrinogen content of the plasma, or at least to observe the clotting time, some three weeks after the diagnosis of fœtal death has been made. This investigation is especially called for if, during the waiting period, spontaneous extragenital hæmorrhage occurs. Evidence of defibrination under these circumstances calls for admission of the patient into hospital for close clinical observation and laboratory control. Spontaneous restoration of plasma fibrinogen concentration presumably will not occur whilst the patient remains undelivered, because there occur both increased utilization of fibrinogen by thromboplastins and increased destruction of fibrin by fibrinolysins. Blood transfusion may be required at this stage to correct any anæmia resulting from hæmorrhagic manifestations.

With regard to the time and method of delivery, it is agreed that, whenever possible, a policy of expectancy should be pursued and the spontaneous onset of labour awaited. There are both advantages and disadvantages in carrying out this policy, and some prefer to take steps to initiate labour by stretching of the cervix and by a carefully supervised œstrogen-oxytocin induction. However, whether the onset of labour is spontaneous or induced, patients in whom defibrination has been demonstrated during the waiting period, on either clinical or laboratory evidence or both, must now have their fibrinogen levels restored sufficiently to bring about stable clot formation, and a minimum of 4 grammes is probably necessary to achieve this. The administration of fibrinogen prior to this time is of little use in restoring the normal blood levels, since this substance continues to disappear from the blood because of the influence of circulating fibrinolysins.

In the presence of prior evidence of defibrination, and in addition to the use of fibrinogen, it is probably wise to maintain a slow blood transfusion during the labour, if only to ensure adequate amounts of available prothrombin. If this is done and fibrinogen levels are maintained, intra-partum and post-partum hæmorrhage need not be anticipated. Moreover, defibrination will not recur after delivery.

Cases V and VI illustrate defibrination and hæmorrhage associated with intrauterine fœtal death. In both instances it was noted that the patient's clinical condition deteriorated more rapidly from the time of onset of the bleeding than would have been expected from the actual amount of blood lost. It has been suggested that this may be due to small spontaneous hæmorrhages into the suprarenal glands.

CASE V.—Mrs. E., a rhesus-negative woman, aged 22 years, pregnant for the third time, had been delivered with her second pregnancy of a hydropic infant. She had shown a progressive rise in blocking antibody titre with this pregnancy, and clinical evidence of intrauterine fœtal death was present from the twenty-fourth week of gestation. After eight weeks of expectant measures, "Pitocin" induction of labour was commenced, and a macerated fœtus together with a degenerating placenta was expelled.

Excessive vaginal bleeding began immediately, the blood did not clot, and the patient's general condition deteriorated rapidly, the systolic blood pressure having dropped to 70 mm. of mercury within a few minutes. Venous blood was taken for a clot behaviour test, and restorative

measures were instituted (administration of "Dextran", followed by whole blood).

Four grammes of fibrinogen were given when, after 15 minutes, it was noted that the blood sample had failed to clot. The response was quite dramatic. Within 20 *minutes of the administration of fibrinogen there was no* further significant vaginal blood loss, and within three-quarters of an hour the patient was again normotensive, *warm and with a good colour and an excellent pulse.* Subsequent progress was uneventful.

CASE VI.—Mrs. F., a rhesus-negative woman, aged 24 *years, pregnant for the second time, was clearly sensitized* to the rhesus antigen with this pregnancy. Clinical and radiological evidence of intrauterine fœtal death was present *from the twenty-eighth week. An expectant policy was* followed throughout, and the patient expelled a small macerated fœtus and a degenerating placenta some seven *weeks later.*

The immediate clinical course was very similar to that described in Case V, and on the basis of a positive result to the clot observation test, 4 grammes of fibrinogen were given by the intravenus route, followed by whole blood. From this stage the clinical course differed in that, although a second sample of venous blood clotted *in vitro* within three minutes, the vaginal bleeding continued unabated, and the systolic blood pressure, which had improved initially, fell again to 80/60 mm. of mercury. During the next hour the patient received a further four grammes of fibrinogen. Vaginal loss gradually ceased and the blood pressure rose to 105/75 mm. of mercury, with a corresponding improvement in the general clinical picture.

SOME OBSTETRIC EXPERIENCES IN MACKAY.[1]

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THIS paper is based on the results of the last 500 cases, in a series of 2000 patients confined in my practice since the war. A previous report (Hopkins, 1955) gave the results of the first 1500 cases of this series. In this paper all tables include the first 1500 cases, shown in groups of 500, for purposes of comparison.

Such statistical results as male, female, types of delivery, parity and Cæsarean section show little change, so as time is limited they have been excluded.

Toxæmia.

The manifestations of toxæmia are shown in Table I.

The toxæmia patients include all those who were admitted to hospital for the treatment of toxæmia, or who developed toxæmia while in hospital. Of the 32 patients admitted for treatment for toxæmia, 15 were primiparæ, and 17 were multiparæ. Eleven of these mothers have had only the one baby so far, 18 have had other babies without toxæmia, and there were three cases of further toxæmia. Of these three, in two the patient was the one mother, who has had five successive stillborn babies owing to toxæmia, and the other mother lost her first baby, which died *in utero* at six and a half months from toxæmia of the mother. One of the two cases of hydramnios was mild, and a living normal baby was born; in the other a grossly abnormal baby was born which died in 20 hours. (This mother has since had a normal baby without toxæmia.)

Of the two chronic nephritics, one was diagnosed by renal function tests six months after the confinement, the other was diagnosed as a nephritic before she became pregnant. Both these patients came through the ordeal fairly well, but they have been advised against further pregnancy.

Eclampsia.

There were three cases of eclampsia in this group of 500 cases. In the full series of 2000 cases, there were no cases of eclampsia in the first group of 500, three in the second group, none in the third group and three in the fourth group. Between the last case in the second group and the first one in the last group there were 679 cases and four years in time. The first three occurred over a period of 12 months, and the next three over a period of 16 months. Six cases of eclampsia in 2000 patients is a lot *by modern standards.*

In a paper on the incidence of eclampsia in general practice in New South Wales and Queensland by the Research Committees of the New South Wales and Queensland Faculties of the College of General Practitioners (1956) *the following figures are given.*

TABLE I.

Manifestations of Toxæmia.	Group. 1 to 500	501 to 1000	1001 to 1500	1501 to 2000	Total.
Hyperemesis	1	1	—	—	2
Hypertension	—	—	1	—	1
Preeclampsia	26	34	33	25	118
Eclampsia	—	3	—	3	6
Chronic nephritis ..	1	1	—	2	4
Hydramnios	—	—	—	2	2
Total	28	39	34	32	133

On the central and north coasts, there were during 1954 six cases of eclampsia in 2681 patients. This area extended from Rockhampton in the south to Cooktown in the far north. In this paper, which covered 1954 only, in all the country areas of Queensland there were nine cases of eclampsia among 4245 patients, so that the number of cases was higher in that year in the central and north coastal areas.

The six patients involved in this report were aged between 22 and 26 years; one case occurred in March, one case in April, two cases in June, one case in August and one case in December; four patients were primiparæ and two were multiparæ.

Four babies were males and two females; one was premature (six and a half months), the others were all born at or about term. All six of these mothers were born and lived in central or North Queensland; three were farmers, or farm workers' wives; three were townsfolk. Four of the six have since had living babies, one having had toxæmia again; this patient was delivered by Cæsarean section. In retrospect, this patient should have had a Cæsarean section the first time; she weighed 18 st. 3 lb. when she booked for the first baby, and it was stillborn, weighing 10 lb. 10 oz. Of the remaining two who have not had babies, one is pregnant at present. Of these six patients, four were admitted to hospital for treatment and induction of labour and still had fits.

The three in the last group were not grossly toxæmic, but they had fits.

In an earlier paper (Hopkins, 1956), a period was covered from January 1, 1945, to December 31, 1954. This paper reported 4652 cases, with 13 cases of eclampsia. This series of cases has been extended to December 31, 1957, and there are now 6542 cases with 15 cases of eclampsia. Five of the six cases previously mentioned are included in these 15 cases. As this is a much larger series, some facts are given for these cases, in the search for causes of the high proportion of eclampsia in this area.

Of these fifteen patients, the ages range from 20 to 36 years. Eleven were primiparæ and four multiparæ. The ages of the multiparæ were 24, 31, 32 and 36 years respectively; three were pregnant for the fourth time and one was pregnant for the third time. Eleven of these babies were males and four were females.

Nine of these patients had ante-partum fits only. I have included fits before and during labour in this category. Three patients had post-partum fits only, and three had both ante-partum and post-partum fits.

[1] Read at the North Queensland Medical Conference, Mackay, September 22 to 29, 1958.

Ten of these patients were country people, and five live in the town. This proportion of two country to one town person is the normal proportion of the population of this area.

This is a prosperous sugar-farming area with a tropical climate, so our patients should all have a reasonable diet. These were all private patients.

Twelve of these cases occurred between April and August, so the winter seems to be the dangerous period, not the hot summer.

Nine of these patients were admitted to hospital for treatment for toxæmia; the other six came into hospital in labour, in toxæmia.

Two mothers died, and three had stillborn babies. One mother (1947) was admitted to hospital in convulsions; she had a Cæsarean delivery of a stillborn child, continued to have fits and died. The other mother had a severe cerebral hæmorrhage with her first post-partum fit and died of this hæmorrhage.

Table II shows the number of cases of eclampsia occurring in each year, with the number of patients confined in that year.

TABLE II.

Year.	Number of Confinements.	Number of Cases of Eclampsia.
1945	308	2
1946	345	0
1947	412	1[1]
1948	438	0
1949	471	3
1950	478	2
1951	512	0
1952	548	2[2]
1953	528	2
1954	612	1
1955	626	1
1956	632	0
1957	632	1
Total ..	6542	15

[1] Patient died.
[2] One patient died.

Three of these patients were treated by Cæsarean section. One mother and baby died, and the others survived; these two mothers having had no fits after operation. The one who died continued to have fits after operation.

Sedation, rest and colon irrigation were used in the earlier years. In 1949 intravenous injections of 50% magnesium sulphate solution and 20% or 25% dextrose solutions were introduced, also restriction of salt intake. After 1949 colon irrigations were dropped from treatment, and in 1952 blood transfusion was used. During the last few years the use of hypotensive drugs has been introduced.

There have been quite a number of patients with severe toxæmia who have been treated in the usual way, and most of these have not developed eclampsia.

During the last few years, some of the cases of eclampsia have started as moderately severe toxæmia; the patients have responded to treatment, and after induction of labour, they have deteriorated rapidly during labour and have had one or two fits. These patients have not, at any stage, appeared to be as ill as some of those with severe toxæmia who did not develop eclampsia.

It is impossible to state, in these 15 cases, what the ante-natal attendances were. Of my own six patients, four were regular attendants, while two attended fairly regularly only. One of these lived 75 miles away and the other 45 miles away; that meant a total journey of 150 miles and 90 miles respectively for each ante-natal visit. Distance does make some difference in ante-natal attendance; however, the two patients mentioned above were fairly good attenders, but did miss a month occasionally, and in the last month an occasional weekly call at a vital time. It must be remembered that we occasionally have patients who come 100 miles.

Most farmers' wives in this area work very hard; some even out cane. I had one patient who did all the cooking and washing for nine men, rising at 4.30 a.m. and going to bed at 10.30 p.m. Another, during one pregnancy cut cane, and during another snigged logs, and quite a number have helped their husbands on the farm, cutting and loading cane. I have followed a number of these patients and this hard work does not seem to lead to toxæmia.

Nine of these patients were admitted to hospital for treatment, and these results should have been better. However, the findings do indicate that they were "picked up" in the ante-natal supervision.

Hospital accommodation is a real problem at times. The hospitals here are very good, taking our patients whenever possible. In considering the admission to hospital of a patient with mild toxæmia, the accommodation factor ruling at the time must be important. The larger of the two private hospitals in Mackay, has "officially" 22 beds, and during 1957 640 patients were confined in this hospital, so that at times accommodation is taxed to the utmost. The new wing to the Mater Hospital will be a very real help to us in dealing with this problem.

The long period of four years without a case, in my series, does suggest a later relaxation of standards of which I was not aware. It would appear that no magic formula is needed to solve this problem. Obviously the needs are the following: (i) more rigorous ante-natal supervision, care being taken that standards are not relaxed with the passage of time; (ii) more meticulous attention to detail in the treatment of preeclamptic patients who have been admitted to hospital for treatment.

Still Birth.

The causes of still birth are shown in Table III.

TABLE III.

Cause.	Group. 1 to 500	501 to 1000	1001 to 1500	1501 to 2000	Total.
Placenta prævia ..	1	—	—	—	1
Hydrops fœtalis	1	—	—	—	1
Abnormality of fœtus ..	2	—	—	—	2
Prolapse of cord ..	2	1	—	—	3
Placenta accreta .	—	1	—	—	1
Toxæmia (maternal) ..	3	3	1	4	11
Maternal dysentery ..	—	1	—	—	1
Cord tight round neck	—	1	—	—	1
Cord tight round elbow and knee	—	—	1	—	1
Suffocation *in utero* of second twin	—	—	—	1	1
Difficult delivery ..	—	—	—	1	1
No cause discovered ..	1	2	3	2	8
Total	1	9	5	8	32

There were eight cases of still birth. Four were due to toxæmia in the mother; two of these cases were from the same mother, who has had five successive still births due to toxæmia.

There is a slight rise in this group, and the figure of 16 per 1000 cases is the same for this group as for the overall 2000 cases.

Neonatal Mortality.

The neonatal mortality and its causes are shown in Table IV.

This gives a neonatal mortality rate of 10 per 1000 cases, and makes the overall neonatal mortality rate 11·5 per 1000 live and still births.

Prematurity is still the greatest cause of neonatal death in this series.

Fœtal Abnormality.

There were eight cases of fœtal abnormality. It seems irrational to allow that if rubella can cause fœtal abnormality, other virus conditions do not do so. I believe that virus and other diseases in early pregnancy should be treated seriously, as they may cause abnormality in the infant. The following is a brief outline of the early history of these eight cases:

1. The baby was a mental defective with a congenital dislocation of the hip. The mother at two and a half to three months' gestation had a hæmorrhage with very septic discharge.

2. The infant had a diaphragmatic hernia and a bowel abnormality and died in the neonatal period. The mother had severe acute tonsillitis at five to six weeks of pregnancy.

3. The infant had congenital heart disease. The mother had a very bad cold at two and a half to three months of pregnancy.

4. The baby had a hare-lip. The mother had pyelitis at two and a half to three months' gestation.

5. The child had an abnormality of the joints of the fingers; the mother had a slight hæmorrhage at two months' gestation.

6. The baby had accessory little fingers on both hands. The mother had impacted retroversion of the uterus of pregnancy; the uterus was replaced.

7. The baby had a meningomyelocele. The mother had had severe vomiting for two to three months—almost hyperemesis.

8. The infant had a cleft soft palate. The mother had severe migraine associated with the diagnosing and treatment of another child with malignant disease. This commenced at about two months' gestation.

Table IV.

Cause.	Group. 1 to 501	501 to 1000	1001 to 1500	1501 to 2000	Total.
Cord round neck	1	—	—	—	1
Prematurity	5	—	2	3	10
Birth injury, cerebral hæmorrhage	—	3	1	1	5
Post-maturity shock	—	1	—	—	1
Atelectasis	—	1	—	—	1
Attenuated cord, baby marasmic	—	1	—	—	1
Abnormality in fœtus	—	—	2	1	3
Hyaline membrane (Cæsarean section)	—	—	1	—	1
Total	6	6	6	5	23

While it is realized these few cases do not prove anything, I suggest that any illness early in pregnancy should be treated seriously.

The greatest value in recording cases such as these lies in the possibility of seeing each case, particularly the difficult ones, in restrospect, after the tumult and the shouting have died away. The following factors emerge:

1. Greater care is necessary in the toxæmias, if eclampsia is to be avoided or reduced.

2. Of the causes of still birth, something is to be learned from reviewing this series.

There were four cases of maternal toxæmia. Two of these were in the same mother, and made the fourth and fifth still births at the same stage with this patient; it is considered that these could not have been avoided. One of the remaining two cases was in a primipara with toxæmia and uterine inertia. There was no great distress, when suddenly at 11.30 in the morning the fœtal heart could no longer be heard. There was no indication for Cæsarean section. The fourth case was one of toxæmia with uterine inertia. This patient did show distress and the baby died *in utero*. As there was no advance over a long period, a Cæsarean operation was performed in spite of the fact that the baby was known to be dead.

A second twin was still-born; an immediate extraction might have saved this child.

There was one still birth associated with a difficult delivery. The patient was to have a Cæsarean section and came into labour a little prematurely. The child was presenting by the breech, and it was decided that the patient should have the child naturally, a wrong decision.

For some still births, no cause was discovered. It is difficult to see any way in which these could have been avoided.

In retrospect, it might have been possible to save two of these eight babies.

3. The causes of the neonatal deaths were as follows. (*a*) Abnormality: this child was grossly abnormal and could not have lived. (*b*) Premature birth: of these infants, one was of less than seven months' gestation and weighed 2 lb. 8 oz., another was at seven months' gestation, one was at a week over seven months' gestation. Possibly with better facilities or care some of these may have been saved. (*c*) Cerebral hæmorrhage: this child was born prematurely at eight months.

Of the neonatal group, the premature babies were the only ones who could have survived. At these hospitals a number of very premature babies have been saved by the care and devotion of the nursing staff. We must all concentrate on the endeavour to save more premature babies, as prematurity is by far the greatest cause of neonatal death in this series.

Conclusion.

In collecting a series such as this, one usually has a salutary lesson. If our figures are to be improved, we must keep a careful check on our results, and reconstruct all casualties, so as to fix them in our memory. This is what is usually called experience; but one must have a clear memory of difficult past cases if one is to benefit by past experience.

Acknowledgements.

I should like to acknowledge a debt to Mrs. Alex. Campbell and Miss Joan Hill for their assistance in keeping these records. I should like to thank all the private medical practitioners in Mackay for permission to use their case records.

EMPHYSEMA.[1]

By R. A. Douglas,
Townsville, Queensland.

> Now, since the muscles of inspiration are numerous and powerful, while expiration, on the other hand, is produced merely by the elasticity of the parts and by the feeble contraction of the intercostal muscles, it must frequently happen that the air, which during inspiration had overcome the resistance offered to its entrance by the tumid state of the bronchial membrane and the sputa, is unable to force the same obstacles during expiration and remains, therefore, imprisoned in the cells, by a mechanism somewhat similar to the valve of an air gun. The succeeding inspirations, or at least such of them as are energetic, introduce a fresh supply of air into the same cells, and thereby necessarily occasion their dilatation and, provided the obstruction is of some continuance, the dilated condition of the cells will be rendered permanent. It follows from this view of the matter that the dry catarrh tends as naturally to the production of emphysema of the lungs as the chronic mucous catarrh leads to dilatation of the bronchia.—Laennec (1829).

It is interesting that modern views on the pathogenesis of emphysema are returning to the views propounded with such perspicacity by Laennec so long ago. I shall

[1] Read at the North Queensland Medical Conference, Mackay, September 22 to 29, 1958.

confine my remarks to the chronic diffuse type of emphysema of the lungs known as chronic hypertrophic emphysema.

The careful studies of Reid (1954) on the pathology of chronic bronchitis have shown that this condition is invariably complicated by emphysema. She is of the opinion, from examination of serial lung sections, that partial inflammatory destruction of the walls of the bronchioles and respiratory bronchioles is often responsible for the formation of flap valves leading to over-inflation and rupture of the alveoli supplied by these bronchioles. Pressure on other bronchioles and minute areas of collapse consequent on the foregoing process tend to produce further emphysema by other inflationary mechanisms. In the condition known as chronic bronchitis, it is the bronchioles which are preponderantly affected by the inflammatory process. All writers on the subject of emphysema, from Laennec onwards, have mentioned the chronic productive cough present for many years, if not the lifetime, of sufferers from emphysema; this, and the chronic or periodic condition of bronchospasm, are features of emphysematous subjects even long before emphysema becomes manifest. Hence it is clear that emphysema is almost invariably an inevitable complication of chronic bronchitis.

Flavell (1952) states that healthy alveoli cannot be ruptured by any physiological range of pressure, so that it is unlikely that the tussive effort itself could be responsible for emphysema, though once the process as described above has commenced, cough could possibly by itself accentuate the inflation already present. Gough (1955) points out that no emphysema can be detected in subjects of pure spasmodic asthma coming to post-mortem examination after status asthmaticus. Hence, pure bronchospasm may be dismissed as a cause of emphysema.

The patient with emphysema presents complaining of a steadily increasing breathlessness, related to exercise and aggravated by recurrent respiratory infections. In the advanced case, the clinical picture is unmistakable, with the barrel-shaped chest and the increased tension in the muscles of the neck and shoulder girdle, all brought into play in an attempt to inflate the already over-inflated lungs. Central cyanosis may be present, together with compensatory polycythæmia. Chest movements are minimal, there is a hyperresonant percussion note, and the vesicular murmur may be barely audible. The cardiac impulse cannot be palpated, and on percussion the heart is overlaid by hyperresonant lung. Sometimes the cervical veins are prominent because of muscular pressure as the neck veins enter the thorax. It is common to find these patients treated as suffering from cardiac failure and not emphysema.

The radiological changes in the chest are due to the increased amount of air in the lungs, as evidenced by the low and flat position of the diaphragm, often best seen in the lateral view of the chest. Sometimes large air cysts can be distinguished; these are only an extension of the emphysematous process on a larger scale. The diminished amount of capillary blood in the lungs accounts partly for the radio-translucency, and the branches of the pulmonary arteries are attenuated as they leave the hilum. The pulmonary arteries themselves are often prominent, owing to increased pressure at the periphery. The heart shadow is small and of the "long drop" type. In the stage of chronic bronchitis before appreciable emphysema has developed, the radiological appearance of the chest is normal.

The electrocardiogram shows a vertical clockwise-rotated heart, and when pulmonary hypertension becomes appreciable, the first abnormality to appear will be the *P* pulmonale, with perhaps later the pattern of frank right ventricular hypertrophy.

As the disease progresses and the vascular bed in the lungs becomes more restricted, the pressure in the pulmonary artery rises, and hypoxia as a result of poor diffusion will further raise this pulmonary arterial pressure. Hypoxia will also have the effect of producing a hyperdynamic circulation, with a high systemic pulse pressure, warm hands with digital pulsation, and engorged cervical veins; œdema of the extremities may also develop, to produce a typical picture of so-called high-output heart failure. By this time a triple cardiac rhythm will be in evidence. At a later stage, as the right ventricle wearies from its struggle to maintain a high cardiac output against the increasing resistance of the lesser circulation, then it will fail, with the production of low-output heart failure characterized by cold extremities and perhaps the development of functional tricuspid valve incompetence.

I have often observed in cases of advanced emphysema, even before cor pulmonale develops, that a patient whose systemic blood pressure was slightly elevated or normal previously suddenly develops severe hypertension, with manifestations of the arteriolar necrosis of malignant hypertension. This has also been observed by Geschichter and Popovicki (1953). Often this variety of systemic hypertension, called phrenic or post-emphysematous hypertension, responds well to bed rest alone, reappearing again when the patient becomes active.

Headache is seen in about 50% of severely affected patients, and is due to dilatation of cerebral blood vessels from carbon dioxide retention. Papilloedema may also be seen.

It is also common to find the complaint of upper abdominal pain, often mistaken for peptic ulcer pain, and presumably due to muscle pain from constant contraction of the diaphragm.

The treatment of emphysema is naturally unsatisfactory in such a widespread destructive process. The most important treatment lies in attempted prevention through treatment of the preceding bronchitis. That this bronchitis is infective in nature has been amply proven from the microscopic pathology of the disease, and from the results of bacteriological examination of the bronchial secretions. It seems established that, when pus is present in the sputum, the responsible organism is *Hæmophilus influenzæ* in about 80% of cases and the pneumococcus in about 20% of cases, according to May (1958). With the appropriate antibiotic treatment relief of bronchitis is remarkable, even if there is a tendency to relapse following treatment. A good case can be made out for continuous antibiotic therapy for the patient infected with *H. influenzæ*. Only by the vigorous antibiotic treatment of chronic bronchitis in its early stages will the incidence of emphysema be lessened. Once the emphysematous process is grossly established, any treatment is merely palliative. Breathing exercises are of some value. When the dyspnœa becomes so severe that life is a burden, pneumoperitoneum will often markedly relieve the dyspnœa and also remove the upper abdominal pain. The effect is obtained by forced elevation of the diaphragm, so that this muscle is again able to contract in a purposive fashion.

When cor pulmonale supervenes, then in the earlier stage, when a hyperdynamic circulation is present, digitalis can be of no benefit, but if it is used with due regard to dosage it will do no harm. However, in the physiological œdema of this stage of the disease, diuretics are contraindicated. When the stage of low-output failure develops, then diuretics and digitalis are indicated. Oxygen therapy should be used, but with due circumspection, as in cases in which the respiratory centre is deadened to the normal stimulus of carbon dioxide and is stimulated only by oxygen tension, then too much oxygen may result in a depression of respiration sufficient to cause coma and death from carbon dioxide accumulation. In a similar way, morphine can be a rapidly lethal agent in severe emphysema. Marked relief of the heart failure will often be obtained by the treatment of exacerbations of the bronchitis with antibiotics and bronchodilators.

Conclusion.

As so often in medicine, the wheel has turned full circle, and we are back to Laennec's views of 1829 on the pathogenesis of emphysema. Probably we are in a

better position now, with our potent armamentarium of antibiotics, to prevent emphysema. However, time alone will tell.

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SOME ASPECTS OF PREVENTIVE MEDICINE IN PÆDIATRICS.[1]

By J. BRODY,

Gordonvale, Queensland.

As a student, I remember reading about the ante-natal care of the child's teeth and thinking it a great joke; today I realize that, far from being funny, this is an important facet of preventive pædiatrics. Indeed, in the pædiatric field we have perhaps advanced further from a curative to a preventive approach than in any other specialty.

I shall touch on some aspects of preventive pædiatrics; it will necessarily be a fragmentary view of an enormous and ever-expanding subject. This subject can be conveniently divided into ante-natal, natal (or perinatal) and post-natal periods; the last-mentioned again can be subdivided into the neonatal (birth to four weeks) period, infancy (one month to twelve months) and so forth.

The Ante-Natal Period.

The modern concept of regular, at least monthly, examinations during pregnancy has done a great deal to lower both the still-birth rate and infant mortality, but more remains to be done. Blood examination early during the pregnancy will reveal, among other things, syphilis (in which adequate therapy will prevent the development of congenital disease), anæmia (which should be quickly and energetically corrected—oral or intramuscular or intravenous administration of iron will usually achieve this), and Rh incompatibility or, rather, the need to watch for it. A thorough history, particularly with respect to previous pregnancies and miscarriages, is, of course, essential, and may provide valuable pointers to possible preventive measures. Adequate nutrition must be ensured, and this may be difficult in the face of persistent vomiting or anorexia; especially the intake of proteins, calcium, iron and vitamins must be watched.

I have found the recording of weight on a graph invaluable in drawing attention to excessive weight gain. Indeed, weight variations are a much more sensitive indicator of impending trouble than variations in blood pressure; albuminuria, of course, is a much later event in the natural history of toxæmia of pregnancy. Undue weight gain can almost invariably be controlled by restriction of the intake of sodium and refined wheat products. If excessive weight gain occurs in the first 30 or 32 weeks of pregnancy, restriction of refined wheat products in the diet is usually the most important therapeutic measure; later, restriction of sodium intake, if necessary with bed rest, is essential. Bed rest, incidentally, means hospital; it is quite unrealistic to expect a busy mother and housewife to stay in bed while she feels perfectly well, just on the say-so of her doctor.

An interesting point, which, however, is only rarely likely to arise, is that there is evidence that excessive intake of vitamin D during pregnancy may cause severe hypercalcæmia (craniostenosis, etc.) in infants. More important, and perhaps not widely enough known, is the vulnerability of fœtal brain tissue to X rays, especially during the first three months of fœtal life. Severe brain damage and microcephaly may result. Routine X-ray pelvimetry therefore must be condemned, and where X-ray examinations are required, they should, if possible, be delayed till after the seventh month.

The Natal Period.

Natal damage (i.e., occurring during labour) may have a number of causes, most of which are well known to most of you. However, let me especially stress the danger of anoxia during labour. Additional oxygen given to the mother during anæsthesia, during the whole of premature labour, and during any forceps or breech delivery, may prevent many cases of brain damage, spastic paraplegia or epilepsy; and oxygen is, after all, so little trouble to administer.

There is no unanimity about the question of post-maturity. I have adopted the habit of inducing labour in all cases after 42 weeks, provided that the duration of the pregnancy is certain. I use surgical induction (rupture of membranes) exclusively, together with the administration of up to eight minims of "Pitocin" in divided doses, and have had no reason to regret it. There is no doubt that statistically there is a higher incidence of morbidity and mortality in post-mature babies, possibly owing to relative anoxia; indeed, a post-mature placenta with extensive calcification and infarction is rather suggestive of anoxia, and to me, convincing evidence.

I search diligently for breech positions during pregnancy, and perform external version if I find any after the seventh month; usually this is easy and does not need to be repeated. I regard every breech delivery as a defeat in my preventive programme.

As a student I was taught that traction on the umbilical cord was the main cause of umbilical hernia. I now believe that infection is the commonest cause. For some years I have been using cord clamps instead of ties; they separate in 48 to 72 hours and leave a clean, granulating surface. The result has been a sharp drop in the incidence of umbilical hernia and a more contented nursing staff—no frequent dressings of foul-smelling cords.

The Neonatal Period.

To turn to the neonatal period, hæmorrhagic disease of the new-born has almost disappeared since the routine use of vitamin K. The life-saving drama of exchange transfusion in Rh or A B O incompatibility is becoming customary, if not commonplace. The discovery of oxygen intoxication with its sequela of retrolental fibroplasia has saved the vision of many premature infants. Much less dramatic, but equally important, are measures such as the routine sucking out of the pharynx immediately after birth—the amount of *liquor amnii* and thick mucus recovered is often quite surprising—and frequent recording of the temperature. Pyrexia due to overdressing in summer is very frequent, at least in the tropics, and can become dangerous. New-born infants, and even more, premature babies having a very limited capacity to sweat and poorly functioning temperature regulation. Pyrexia in neonates, indeed, is commonly an expression of hyperthermia due to environmental factors; changes in respiration, anorexia, vomiting and change in colour are much more reliable signs of infection at that age. An unnecessary bunny rug can send a baby's temperature to 104° F. in a short time (with corresponding return to normal on removal of the offending rug), while massive pneumonia may cause a rise of perhaps no more than from 98° to 99° F., or even a drop to subnormal temperatures.

I shall not deal with the premature infant in this context; in itself this problem presents an enormous field for study, and other than the prevention of prematurity as such, the approach is perhaps more curative than preventive.

[1] Read at the North Queensland Medical Conference, Mackay, September 22 to 29, 1958.

A most important step in the preventive programme is the thorough examination of the baby before he leaves hospital. The baby should be completely undressed, and if a routine order is established in these examinations (head, fontanelles, chest, heart, clavicles, spine, abdomen, hips, feet, etc.), examination need not take more than a very few minutes. An active Moro reflex, equal on both sides, will prove the absence of damage to clavicles, humeri or brachial plexus. However, absence of heart murmurs does not prove freedom from congenital heart lesions, as these murmurs often develop later in life. The sequence of no murmur—systolic murmur—machinery or to-and-fro murmur in patent ductus arteriosus is well known and frequently met with. Weakness or absence of the femoral pulse indicates coarctation of the aorta.

Foot deformities, especially the milder forms of pes varus, pes adductus, metatarsus varus, etc., may be difficult to recognize; familiarity with the appearance of the normal foot of the new-born infant and its normal range of movement will be a great help. Congenital dislocation of the hip is often missed in the early months of life; yet treatment before walking is essential for a good result. Here it is important to see that both hips are equal (posteriorly, inequality of the gluteal folds may be a pointer) and that the thighs move equally and freely to an abduction of about 80° from the mid-line, with the baby on his back, knees fully flexed and thighs flexed to 90° at the hip. Ortoloni's sign (an audible or palpable click on abduction) is an almost certain indication of congenital dislocation, produced by slipping of the head of the femur over the shallow acetabular rim.

Raised hæmangiomata should be treated early when a good cosmetic result is important, as they occasionally tend to enlarge, though most will disappear during the first year of life. A good forceful urinary stream, once seen, will dispel any worry about posterior urethral valves which, untreated, can be the cause of serious renal invalidism due to back pressure.

Of course, such an examination may save you a good deal of embarrassment as well. When the mother asks you about the baby's sixth toe or the spot on his back, it is surely much more dignified to be able to say "I have seen it and it is nothing to worry about", rather than "I didn't know, but I shall have a look".

Active immunization is here to stay, and today is a universally accepted procedure. However, it is necessary to arrive at some compromise between the optimum immunizing effect and the maximum interference tolerated by parents or children. Obviously, a slightly less effective immunizing procedure involving fewer injections, which is completed in 95% of the child population, will be vastly superior to an ideal immunizing programme demanding more attendances and many more injections, which is completed in, say, only 50% of the child population. A useful immunizing schedule, which seems to be acceptable to most mothers and children, is as follows: triple antigen (diphtheria, pertussis, tetanus) at three, four and five months; Salk vaccine at six and seven months; triple antigen or diphtheria and tetanus "booster" at 18 months; Salk vaccine "booster" at 18 months; diphtheria-tetanus "booster" at five years and ten years. This is a total of nine injections over 10 years, but five fall into the first seven months of life and will not be remembered by the child. This schedule will give a good immunity level to four important infectious diseases. However, if the last tetanus injection was given more than, say, six months earlier, and also in the case of a badly contaminated wound, a "booster" dose of tetanus toxoid (not anti-tetanus serum) should be given for any suspect injury.

If this schedule is carried out, only occasional cases of pertussis and diphtheria will occur, and these can be expected to be mild; indeed, I cannot recall a confirmed case of diphtheria in my (admittedly rural) area for over 10 years. Of course, immunization must be "sold" to the parents; the mother during her stay in hospital after confinement will usually be found to be interested and receptive. She should be reminded again to bring her baby for immunization at the appropriate time when she returns for her post-confinement examinations. Immunization propaganda in the schools is also very useful.

Universal vaccination against smallpox is not generally accepted in this country, but in my opinion would be most desirable. With modern air transport, introduction of smallpox to Australia could occur in spite of apparently adequate precautions, and could assume alarming proportions.

Infancy and Early Childhood.

Lastly, let me make a plea for regular, say six-monthly, examination of well children. Toddlers' clinics are conducted by the Department of Health in Brisbane and several country centres, and they have proved immensely popular. They afford an opportunity not only to discover the occasional major disease, but to advise mothers about minor troubles which they are reluctant to take to their own doctor as being too trivial, but which worry them nevertheless. A few words of advice about feeding or behaviour problems, bowel habits or speech peculiarities, or merely telling the mother that her child is doing well, will materially assist her peace of mind and her confidence, and will thus contribute to emotional stability in the home and ultimately to better mental health in the child. Again, this regular visit is an opportunity to check on immunization, to remind the mother when an injection is overdue, and to explain when necessary the importance of immunization. Advice on nutrition, emotional problems, enuresis and umbilical hernia is often requested and usually can be given in a few words, although occasionally a longer session is needed. In the Department of Health's well children's clinics, of course, all treatment and problems requiring more detailed advice are referred to the family doctor; as the name implies, the clinic deals only with well children.

Conclusion.

Time has permitted only a short glimpse into the realm of preventive pædiatrics, an extremely important and ever-increasing subject, and one which is particularly rewarding, even if the results are sometimes not immediately visible or may have to be gleaned from the statistician's reports. I started with a reminiscence; permit me to close with another. Twenty years ago, at the Brisbane Children's Hospital, there was a large ward filled with children suffering from diphtheria, many with tracheotomies or endotracheal tubes, many critically ill, and not a few dying from their disease. The ring of the dreaded tracheotomy bell was a common occurrence, and the signal for the resident to drop what he was doing and to run as fast as he could to the emergency theatre to perform a tracheotomy on a limp, deeply cyanosed child, without taking time for detailed examination or anæsthesia. Indeed, some of you will remember what I am describing. It is a safe bet that today few students in Brisbane have seen a case of diphtheria, and the diphtheria ward has long since been converted for the use of children suffering from other complaints—quite a dramatic change, and one of the many triumphs of preventive pædiatrics.

INTERNAL DRAINAGE OF THE COMMON BILE DUCT.[1]

By L. I. Burt,
Brisbane.

Internal drainage of the common bile duct may be achieved by creating an internal fistula between common bile duct and duodenum. This procedure has been described as a choledochoduodenostomy, and consists of a side-to-side anastomosis between duct and duodenum.

It was mentioned in a previous paper (Burt, 1953) that this subject had been neglected in British surgical literature. It has been pointed out that British surgeons

[1] Read at the North Queensland Medical Conference, Mackay, September 22 to 29, 1958.

were not convinced that recurrent cholangitis with biliary crises would not result. That episodes have been completely absent is, in part, the result of technique, and in part due to local mechanics. Probably, in the past, jaundice and fever have been the result of an obstruction, or have followed cholecystgastrostomy. That these complications have not occurred, and that drainage has been successful, have been shown by the case records.

The Operation.

The operation is described first, so that the indications, implications and possibilities may be more readily appreciated. First it may be stated that the procedure will rarely be called for, except in patients who have a dilated common bile duct.

An incision is made in the common bile duct: not transverse, as this may not allow for a large enough aperture; not longitudinal, as described by Horsford (1958), since this may require pulling up of the duodenum with some tension; but obliquely. The line of the incision runs from left, medially and above, to the right laterally and below, parallel to the upper outer angle of the duodenum, and in that part of the common bile duct immediately above or just posterior to the duodenum. The length of the incision should be maximal, so that, being oblique, it can equal at least half the circumference of the common bile duct. The incision in the duodenum is made parallel to that in the duct, so that the two openings fall naturally together. In length it should be equal to that in the duct. The mucosa pouts out in the form of a rosebud and becomes more plentiful than could be desired.

The common bile duct may be explored and irrigated. The duodenal incision is too superior for visualization of the ampulla; but a forefinger may palpate this region through the opening and a probe may be manipulated through. In this manner patency may be established and a carcinoma palpated. The anastomosis is performed with interrupted sutures closely applied to avoid leakage, and picks up mucosa only of the duodenum and the full thickness of common bile duct until the angles are reached, when it is completed anteriorly by picking up full thickness of both. It will be appreciated that duct and duodenum are in contact inferiorly, so that the first posterior line amounts to the "second row" of the anastomosis. Anteriorly, the second suture line can be effected by union of the peritoneum. Continuous suture is avoided because of purse-string effect and consequent narrowing. Silk has been used, but can be troublesome in an infected field. Size 0 catgut or chromicized gut is preferred.

The Indications for the Operation.

The indications for the operation may be briefly stated as follows: (i) biliary calculi; (ii) pancreatitis with obstruction; (iii) carcinoma of the head of the pancreas: (*a*) palliative, (*b*) first stage of a two-stage procedure; (iv) stricture.

Discussion of the Indications.

Biliary Calculi.—At first, in my own hands, this operation was reserved for patients who had previously had a cholecystectomy and/or exploration of the common bile duct performed, and presented with common duct calculus.

Even a successful clearance and drainage of a common bile duct, the site of a recurrent calculus formation, and dilated, thickened and inflamed, is not a guarantee against the possibility of further calculus formation. Drainage of a gall-bladder provides only temporary relief, and drainage of such a duct may provide relief for only a similar time. Permanent drainage, however, provides more chance of a permanent cure, and overcomes to a large extent not only the formation of new calculi, but the dangers inherent in an overlooked calculus. This latter may pass through the internal fistula.

The operation has been performed on a patient who has had gall-bladder and hepatic and common ducts packed with hundreds of minute cholesterol calculi, each final irrigation producing eventually one more calculus, so that it was thought that calculi would almost certainly be left.

The operation has also been performed on a patient who had pure pigment deposition in the common duct, when it was thought that the cause of this was excess pigment formation as in hæmolysis. I am now inclined to perform the operation on all patients who have dilated ducts owing to obstruction by calculi. It has not been performed when exploration of the duct has shown this to be not dilated and the source of calculi is believed to be the gall-bladder—i.e., minimal abnormality of the duct.

Pancreatitis.—The first case in which I performed this operation ten years ago will demonstrate its use here.

The patient was a middle-aged man, who presented with a history of about two weeks' icterus following an attack of pain. He was green with jaundice. Exploration revealed an apparently normal gall-bladder except for a very mild degree of cholesterosis and two minute cholesterol calculi, and a dilated bile duct containing only some white bile. His liver had apparently ceased secreting. The head of the pancreas was enlarged and hard, and a probe could not be passed into the duodenum. His condition precluded pancreatic biopsy, and carcinoma could not be excluded. A choledochoduodenostomy was performed as described. The gall-bladder was drained, and bile appeared in 48 hours. Subsequent laparotomy revealed that the pancreatic enlargement was due to inflammation. The gall-bladder was removed. The fistula was demonstrated to be patent five years later, and he has enjoyed good health to this day.

Whether there is any advantage to be obtained in chronic or recurrent pancreatitis by providing free drainage of the pancreatic ducts by circumventing the intermittent physiological obstruction of the sphicter of Oddi by this means is not certain, although there is evidence that this may be of help. This is probably true only in those cases due to disease of the biliary passages.

Carcinoma of the Head of the Pancreas.—As a palliative procedure, choledochoduodenostomy has been found useful in allowing patients to die of secondary deposits but without jaundice. For those few patients who have been fortunate enough to have a slowly growing malignant tumour, this has been worth while. The comments of Professor I. Aird in his latest volume, "Companion in Surgical Studies", are instructive in this regard. As a first-stage procedure before pancreaticoduodenectomy, it provides for a quick return to normal physiology without loss of length of common bile duct, since the anastomosis can be excised with the pancreas and duodenum.

Stricture.—The application of the operation here is obvious; nor does the site of the stricture matter, so long as there is sufficient common duct available. The duodenum can be mobilized to reach as high as may be required.

Mechanics and Physiology of the Duodenum.

To be discussed now are the following features, together with comments on the mechanics and physiology of the duodenum: (i) the danger of leakage; (ii) the danger of ascending infection; (iii) the possibility of calculus or food stagnating in the retroduodenal cul-de-sac; (iv) the permanency of the fistula.

The Danger of Leakage.

Leakage could be described as a combined biliary-duodenal fistula, and is a possibility. This can only be discussed on the bases of precautionary measures, incidence and results.

Precautionary Measures.—When dissatisfied with the closure for technical reasons, I have placed a narrow tube to drain the common bile duct above the anastomosis. This has been done in two cases only. Also, a duodenal tube with continuous aspiration has been used for 48 hours. In all cases the region has been drained to the exterior with a tube split for one and a half inches, but not as far as the abdominal wall.

Incidence.—In one case only has there been evidence of a fistula to the exterior, and this closed in about two days after removal of the drainage tube.

Results.—The frightening loss of fluids and electrolytes which can result from a duodenal fistula does not appear

to apply here. Probably such a loss is dependent upon two factors: (i) obstruction, partial or complete, below the fistulous opening; (ii) a fistulous opening in the vicinity of or below the ampulla of Vater. When one is performing a gastrectomy, if the duodenal stump closure is difficult because of fibrosis, a fine tube may be placed in the duodenum and closure made tight around it. This tube may be slowly removed over three days, starting on the third day, and almost no drainage will result.

The Danger of Ascending Infection.

First, this has not occurred.

One patient, a married woman, had had a cholecystectomy performed 14 years before presenting. In the previous 12 months only she had had attacks of pain, fever and jaundice. Operation revealed a dilated common bile duct filled with pure pigment calculi of staghorn variety. Choledochoduodenostomy was performed. All was well for three months, when the patient suffered not colic, but pain and fever without jaundice. There were several attacks, which responded to antibiotics. The common bile duct was outlined by air, and no outline could be obtained by cholangiography; this indicated that the fistula was patent. Subsequent investigation revealed a calcified shadow in the superior portion of the right lobe of the liver, and an immediate positive response to the Casoni test. It is suspected that she has a hydatid cyst of the liver.

No other patient has so far had any symptoms referable to the biliary system.

The Possibility of Calculi or Food Stagnating in the Retroduodenal Cul-de-sac.

This appears to be a definite danger, and could prove to be a factor in limiting the decision to perform the procedure. So far, the evidence is that it does not occur. In most cases the drainage of bile by the normal route is still going on, so that the area drained by the common duct can be drained through both fistula and ampulla. Duct calculi have a habit of moving in the common duct in the manner of a ball valve, and may pass through the fistulous opening.

In an attempt to investigate this problem, four patients were subjected to barium meal X-ray examination. The object was to force barium into the common duct and make further X-ray examinations at intervals. This is a difficult procedure, and more often fails than not. However, in two cases it was successful. In one, there was no barium in the duct within 48 hours, and in the second, a little remained to be seen on the second-day film. In this latter case, the next X-ray film taken on the fourth day showed complete clearance. It will be appreciated that such an investigation requires much time and patience, and to be successful may have to be repeated several times. The duodenum is normally empty and passes its contents rapidly onwards. This fact makes for ready drainage. In a cholecystgastrostomy, considerable intragastric pressure can be built up and may fill the gall-bladder with gastric contents, so resulting in ascending infection. I am sure this does not occur in choledochoduodenostomy, and little food, if any, ever enters the common duct. Were it to do so, it would not remain.

The Permanency of the Fistula.

I believe that this fistula is permanent. The longest time interval in any one case investigated is 10 years. There are several patients who have a proven fistula patent after a period of five years.

A fistula tends to remain patent if there is an obstruction below it. The sphincter of Oddi constitutes an intermittent physiological obstruction. Furthermore, it may be noted that patients who suffer an attack of gall-stone obstruction of the small bowel do not suffer from cholangitis afterwards, and indeed do not again present for biliary surgery. It has been possible to demonstrate the persistence of the spontaneous cholecystduodenostomy years after. Such a method had been used surgically in the treatment of biliary disease in Italy.

In a gastro-enterostomy, in which the pylorus is patent and duodenal stenosis subsides, the stoma tends to narrow. The contribution to this is not from the mucosa, but from fibrosis in the serosa and muscularis. The stoma of the choledochoduodenostomy involves little muscularis and serosa, and is much larger in relation to the diameter of the structures involved.

Investigation of the Patency of the Fistula.

The patency of the fistula can be investigated radiographically in the following way. First, a plain X-ray examination may reveal that the common bile duct is outlined with air or has air bubbles in it. Several films on several days may be necessary to reveal this. If air bubbles are present, they may not be recognized as such, but mistaken for calculi. However, subsequent films may show that the shadows have gone. Second, cholangiography may be employed. In no case of a patent fistula will a satisfactory picture be obtained, since the material moves too fast into the duodenum. This is taken as negative evidence of a patent fistula. Finally, a barium meal X-ray examination is the most convincing way to demonstrate the stoma and the behaviour of material in the termination of the common bile duct. Unfortunately, the attempt may not prove successful.

Conclusions.

This procedure, in spite of original fears and opposition, is coming more and more into use, and in my opinion will have a recognized place in the surgery of biliary disease.

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GALL-BLADDER DISEASE: SYMPTOMS AND DIFFERENTIAL DIAGNOSIS.[1]

By I. A. Lester,

Cairns, North Queensland.

Disease of the gall-bladder is one of the commonest met with in surgical practice. Much has been written about it, especially about the more abstruse aspects and the complications of surgery. In this talk I intend to deal mainly with the everyday problems of the diagnosis and management of common gall-bladder disease. In effect, this means acute or chronic cholecystitis, with or without calculi and the direct or obstructive results of calculi in the gall-bladder itself or the bile ducts.

The gall-bladder is a hollow muscular organ, a foregut derivative with foregut innervation lying in the right upper abdominal quadrant. It is supplied by a single artery, and filled and emptied by a single narrow, tortuous duct which drains into the hepatic duct system. This duct and artery system are the site of numerous variations. The gall-bladder is closely related anatomically to the duodenum and colon. Its function appears to be to concentrate the bile, store it and discharge it into the duodenum as required. Its functions are under combined nervous and hormonal control.

These well-known facts determine to a great extent the nature of the diseases which affect the gall-bladder and the symptoms they produce.

Symptomatology.

I propose only to comment on some aspects of history taking and investigations.

The family history may be helpful in a general way only. I have found it so in the investigation of the unusually young patient with obscure symptoms.

The patient's age also is helpful only in a general way. Gall-bladder disease has been reported at all ages, from the neonatal period onwards, but is is usually a serious consideration in differential diagnosis only

[1] Read at the North Queensland Medical Conference, Mackay, September 22 to 29, 1958.

after the age of 20 years. The youngest person operated on at the Cairns Base Hospital in the last few years was aged 16 years, and the oldest 76 years.

The duration of symptoms also varies enormously. Some patients present with their first attack of colic and deny any previous symptoms. Others give a history dating back as much as 20 years. Long remissions between attacks are also common. One of my patients claims to have been perfectly well for 10 years between two severe attacks of colic.

Physique and parity, too, are helpful only in a general way. Most patients with gall-stones are well covered, and a goodly proportion have borne children. Many, however, are slight in build; I am reminded of a nurse, aged 21 years and weighing only six stone, who had gall-bladder disease of long standing.

History taking should be careful and accurate, and should include an assessment of the severity of the pain. A question as the whether morphine has ever been required is very helpful if one suspects that a patient is exaggerating. Physical examination should also be full and should include all systems, particularly the cardio-vascular system which is often affected in a patient who has gall-bladder disease. Slight tenderness in the right upper abdominal quadrant is often the only positive finding between "attacks". A gall-bladder palpable through the abdominal wall usually indicates disease outside the gall-bladder. Occasionally in a thin patient the gall-bladder, tightly distended by stones, can be easily felt, but its nature is, or soon becomes, obvious.

Ancillary Investigations.

I should next like to make some comments about ancillary investigations; the most important of these, of course, is radiology.

Flat X-ray films are not very helpful by themselves; they must, of course, be taken as a preliminary to cholecystography, otherwise a calculus will occasionally be concealed by the medium. In general, I much prefer oral to intravenous cholecystography, except when the gall-bladder is non-functioning or absent, and when further information is required about the duct system. I have found intravenous cholecystography much less reliable for general purposes. Two important positive findings may be made: (i) if one can be confident that the patient has taken and absorbed the medium, and if the liver is functioning normally, I consider that non-filling is a very reliable indication of gall-bladder disease; (ii) stones may be seen either as positive shadows or as filling defects in the medium. Once again, if one can exclude overlying gas shadows in the bowel or underlying kidney stones, the examination is of great value. Oblique views are very helpful in removing causes of confusion. Of minor abnormalities in the cholecystogram I am most suspicious. In particular, I refer to incomplete emptying and apparent kinks. I have not yet had a case in which these findings were significant. Poor concentration should be looked on with some suspicion, although, of course, it may be significant in some cases.

Liver function tests are mainly of value when jaundice is present, when they probably should always be performed. They are no substitute for an accurate history, but may well confirm the clinical diagnosis and thus increase the surgeon's confidence. The fallacy of the obstructive picture often seen in chlorpromazine jaundice is now well known and must be remembered. The difficulty is that many patients taking chlorpromazine must also have gall-stones.

Blood investigations may be indicated in special cases, and certainly the patient's hæmoglobin value and blood group should always be known before any abdominal operation is undertaken.

Differential Diagnosis.

Gall-bladder disease may be symptomless, or may manifest itself in the following four symptom groups, in any order or combination.

Biliary Colic.

This is the most dramatic. I would remind you that the pain may be central and not felt in the right upper quadrant at all. It is often very severe and accompanied by a marked degree of collapse. It has to be distinguished from such conditions as right renal colic, intestinal colic, perforated peptic ulcer, pancreatitis and severe angina or coronary occlusion. Most of these can be excluded by remembering them and by taking an accurate history. Occasionally ancillary investigations may be needed — for example, a vertical X-ray film of the abdomen to reveal gas under the diaphragm, serum and urine amylase tests or an electrocardiogram may be indicated.

Recurrent Cholecystitis.

The patient may have attacks of cholecystitis recurring at irregular intervals. If there are no other indications of gall-bladder disease, difficulty may be experienced with some of the following conditions.

Duodenal Ulcer.—It should be remembered that this is often coexistent, but a careful history will do much to exclude it. Occasionally, barium meal X-ray examination and cholecystography are necessary, and in any case few surgeons would operate in either condition without X-ray confirmation.

Hepatitis.—I have seen a number of cases in which hepatitis has been misdiagnosed as cholecystitis and operation recommended. This is mainly due to inadequate history taking; but an occasional case of subacute hepatitis with exacerbations in which jaundice, abdominal pain and tenderness occur, may be very misleading. Moreover, these patients will be found on cholecystographic examination to have a non-functioning or poorly functioning gall-bladder. Liver function tests will be decisive if one's suspicions have been aroused.

Pyelitis, Hydronephrosis, Appendicitis and Liver Congestion.

These occasionally cause difficulty, particularly a high appendix. But the patient with really acute inflammation of a high appendix becomes more ill and has a typical "intestinal" breath and tongue, and in any case will usually demand operation eventually. The attacks of liver congestion with a tense, tender liver in early congestive cardiac failure may also simulate recurrent cholecystitis; but a careful history and examination should exclude this dangerous misdiagnosis.

Biliary Dyspepsia, Coronary Artery Disease.

Under this heading one must differentiate all other causes of dyspepsia by a careful history and examination and, when necessary, ancillary investigations. I would especially draw attention to the dyspepsia of overeating, which often coexists with gall-bladder disease, but which will not be cured merely by removing the gall-bladder. In this connexion a period of weight reduction for the obese patient serves the double purpose of making the operation easier for the surgeon and demonstrating to the patient to what extent her symptoms are due to her dietary habits. Coronary artery disease may also simulate dyspepsia and, indeed, often coexists with gall-bladder disease. Its presence must always be considered and watched for.

Jaundice.

The differential diagnosis of the cause of jaundice is a subject to itself, and it is impossible to discuss it in this talk. Carcinoma of the pancreas and the related ampullary carcinoma is the commonest item to consider. It is now well known that jaundice due to this cause is not always steadily progressive or painless; nevertheless, in the great majority of cases the diagnosis is straightforward, clinically and with the aid of ancillary methods. In the case of mild transient jaundice, hepatitis, as was previously mentioned, must be thought of and ruled out. Chlorpromazine jaundice also has already been mentioned, and a specific inquiry as to whether the patient is taking any drugs must be made. In most cases of persistent obstructive jaundice, laparotomy will be indicated and is the final court of appeal.

Post-Cholecystectomy Syndrome.

I do not know whether the post-cholecystectomy syndrome is strictly relevant in the discussion of gall-bladder diseases; however, I propose merely to remind you that it may be due to the following conditions in the order of probability — (i) a missed or recurrent stone; (ii) injury to the bile duct; (iii) fibrosis of the ampullary region; (iv) chronic pancreatitis; (v) misdiagnosis of the original condition, especially a peptic ulcer. Perhaps this last item should have been put far higher on my list.

Indications for Treatment.

It would probably be admitted that medical treatment (except dietary treatment and especially weight reduction) is of value only in the relief of acute symptoms. If this admission is made, discussion of the treatment revolves around a number of questions.

1. Should all gall-stones, even symptomless ones, be dealt with surgically? This question can always be relied upon to produce a good deal of argument. When one realizes that a gall-stone may lie silent for many years and then cause a dangerous complication, it seems probably correct to recommend cholecystectomy for all such patients; it must be hastily added that common sense will dictate when elderly patients and patients who would be "poor operative risks" must be excluded, and that the decision applies only when really competent surgery and anæsthesia are available. The common bile duct area provides too many pitfalls to be the playground of the inexperienced. However, before removing the silent gall stone, one should be careful to explain to the patient that the operation is being carried out as an "insurance" measure, and that it is unlikely to alter irrelevant symptoms.

2. The next question is when to operate in a case of acute cholecystitis. In the enormous majority, of course, the condition settles without interference. In those in which intervention is required, the need for this soon becomes fairly obvious; the patient's condition is not settling, and his abdominal tenderness and rigidity are spreading. In most of these cases, it will be found that perforation has already occurred into a localized area walled off by omentum and surrounding organs. In my opinion, operation should almost always consist in simple drainage of the gall-bladder, as cholecystectomy in these cases introduces considerable extra risk and achieves only a gain of a few weeks of the patient's time. In a few cases in which the duct system is not involved in the inflammatory process, cholecystectomy by a very experienced operator would be justifiable.

3. When to operate on a jaundiced patient? The great majority of patients with stone obstruction settle down and can be operated upon when not jaundiced. It is important not to wait too long, or another attack may supervene. In the case in which the condition does not settle and the patient remains jaundiced, the diagnosis will often be in doubt even after the most intensive investigations. If the jaundice has been proved to be obstructive, laparotomy should be carried out to make a diagnosis and deal with the cause of the condition. Jaundiced patients, on the whole, rarely present any real problem in decision.

4. The patient with the normal cholecystogram who has typical gall-bladder symptoms, however, often causes a great deal of difficulty. My experience has been that operation in these circumstances rarely produces a grateful patient. I have operated only on four or five such patients in the last few years. Some claimed to be temporarily better after the operation, but only one has received any apparent permanent benefit.

This was a trainee nurse, who was admitted to hospital on a number of occasions with severe right upper quadrant pain and tenderness and severe vomiting. Her training was being seriously interfered with; but investigations of her gall bladder and stomach gave persistently normal results. She was otherwise a sturdy, healthy type, both physically and mentally. After discussion with the girl and her parents, it was decided to remove the gall-bladder. Since the operation she has had no further trouble, and has completed both her general and midwifery training, so that the follow-up period has been reasonably long. Nor have any replacement neurotic systems come to light.

In spite of this case, I would repeat that operation in this type of case rarely produces a happy result. In most cases I believe the patient's symptoms are due to "nervous dyspepsia", whatever that may be.

Conclusion.

In such a common disease, it is important that the surgeon should have a clear idea of what he is setting out to achieve. I have tried in this talk to discuss some of the points which have caused me difficulty with my own cases. The lesson to be learned is, I think, the old one — that a carefully taken history and a carefully conducted examination are the most important single factors in doing the best possible for the patient. An accurate knowledge of pathology and careful surgery come next. It is also important to treat the patient as a whole. If he is regarded as an organism which happens to be carrying gall-stones, results will be less than perfect.

Acknowledgement.

I wish to thank the Director-General of Health and Medical Services for permission to publish this paper.

Reviews.

Fundamentals in Cardiology. By John B. Wild, M.D.; 1958. Oxford: Blackwell Scientific Publications. 9¼" × 6", pp. 96, with many illustrations. Price: 34s.

Since the time when cardiac catheterization established itself as a routine procedure in the elucidation of the diagnosis and hæmodynamic mechanisms of cardiac disease, there has been an increasing need for a small monograph on the basic pressure curves from all parts of the heart, and for the correlation of these with electrical, audible and visible phenomena of the cardiac circle. Many such relationships have appeared in the classical physiological contributions of Wiggins, Bramwell and many others, but they have chiefly been derived from animals rather than man, and a study of these phenomena involves a wide search of the literature.

Dr. John Wild has recently produced an excellent summary of the mechanical changes in intracardiac pressures as recorded through catheters in the right and left cardiac chambers, both in normal hearts and in those affected by congenital or acquired valvular disease. By serial imposition of one tracing upon another, he has made the various interrelationships much easier for the medical student to understand. The text is simple, direct and concise, so that each diagram is situated close to its textual description, and the figures rather than the words form the main teaching medium. The interpretation of clinical findings, from waves in the neck veins to additional diastolic sounds, is made much easier and more interesting for anyone fortunate enough to have read this book simultaneously with their tutorials in physical diagnosis. Dr. Wild has emphasized principles and exactitude, and has created a very valuable and illuminating adjunct to the demonstrations of the bedside tutor, and to a study of tracings and phonocardiograms from the cardiac laboratory. Interest in the absorbing subject of clinical signs and their physiological interpretation will be perceptibly intensified for anyone who reads this little book attentively.

Hearing Therapy for Children. Edited by Alice Streng, M.A., W. J. Fitch, M.A., LeR. D. Hedgecock, Ph.D., J. W. Phillips, M.D., J. A. Carrell, Ph.D.; Second, Revised, Edition; 1958. New York and London: Grune and Stratton. 8½" × 5½", pp. 360, with many illustrations. Price: $6.75.

The title of this book reflects the changing attitude to the development of communication skills in children with hearing defects, emphasizing the rightful heritage of all to make use of any hearing potential they have. Pursuit of this objective entails excursions into methods of audiometry and the anatomy and physiology of the hearing apparatus, as well as auditory training and educational procedures considered by the authors as applicable. Their choice could, however, provide cause for contro-

versy in the last-mentioned field. The multiplicity of authorship is betrayed by some strange contextual placing of valuable, perhaps even vital, scraps of information. At times the discourse rambles off into some aspect of the subject of undeniable value, but not appropriately placed.

This attempt at a comprehensive survey of the subject is not new. Although the book can hardly be said to be true to its title, the authors have collated all the material of practical value in providing a full oral life for those with hearing defects, according to the highest accepted standards in U.S.A. today. This is an acceptable contribution for pædiatricians, general practitioners, and therapists working with speech defectives as well as those deprived of hearing. To it as an encyclopædic survey, the parents of a deaf child can refer in their search for those basic principles which make understanding of their child's problem more complete and their personal contribution to his development of language and speech more effective.

Treatment of Breast Tumors. By Robert S. Pollack, M.D., F.A.C.S.; 1958. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 10" x 6¾", pp. 148, with 47 plates and 16 text figures. Price: 66s.

THIS small volume presents a very good over-all survey of tumours of the breast, with most of its emphasis on the subject of carcinoma and its management. The author has had the assistance of several of his Californian colleagues in the preparation of chapters on ancillary methods of treatment, such as radiotherapy, hormonal control, adrenalectomy, hypophysectomy and superradical surgery. These special chapters, while brief, help to make the volume much more useful. The general presentation is good, and the diagrams and illustrations are excellent and numerous. The author is a surgeon of experience and mature judgement, and it is perhaps fair to say that the chapters written by himself are the most impressive ones. The pathological classification is not one in wide general use, but can be correlated with other systems. No reference is made to metastatic tumours in the breast, and Paget's disease receives scanty mention, particularly by comparison with cystosarcoma phyllodes. Few surgeons in this country would favour prophylactic amputation of the contralateral breast in cases of carcinoma, as the salvage would not justify the mutilation. The discussion of the indications for the use of radiotherapy in conjunction with surgery is good and reflects sound understanding of the disease and its management. This volume can be commended to those who are interested in diseases of the breast at specialist or general practitioner level.

Practical Biology for Advanced Level, Medical and Intermediate Students. Volume II: Practical Botany. By C. J. Wallis, M.A.; Fourth Edition. London: William Heinemann (Medical Books), Limited. 8½" x 5½", pp. 186, with 71 illustrations. Price: 17s. 6d. (English).

THIS book is clearly printed and nicely bound, with useful appendices on methods and reagents, and few observed misprints. The inclusion of bacteria in a practical botany course is good. Amongst desirable information on microscopic technique is much that is excessive or too complicated for first-year university or final-year school botany or biology in Australia. This trend to complication or impressiveness is exemplified by the extra formula (page 9) for magnification with extended draw-tube, when it is necessary only to change one value in the original formula. In the section on "Elementary Biochemistry", the unexplained use of complicated compounds leads, at this level, to learning by rote. Undesirable are chemical reactions in molecular formulæ without algebraic balance—for example (page 34, with probably a misprint or two):

$$C_6H_{10}O_4(H_2PO_4)_2 + H_3PO_4 \rightarrow CH_3.CO.COOH.$$

Up-to-dateness should be accompanied by correct usage. Originally introduced for flexibility and avoidance of rigid derivation of tissues under the histogen theory, the concepts of *tunica* and *corpus* are not only wrongly used, but have their very purpose transgressed in the paragraphs on apical meristems on page 89 and 96.

Despite the author's *caveat* (page vi), "many of the illustrations in this manual are intentionally diagrammatic or semi-diagrammatic . . .", the details of a figure should be correct as far as they go. "Pebbly beach" and "rice pudding" tissues are abhorrent. The student is misinformed by Figure 46 of *Tilia* stem showing rounded phellogen cells, cork cells in rows not conforming with them, and the secondary phloem cells without differentiation or regularity. The *Cheiranthus* illustrations (Figure 51) contradict one another in the ovary, and the figure illustrates flower structure described for *Ranunculus*.

An unfavourable review should be supported amply by detailed reference. The following, culled from the many possible examples, demonstrate the book's erroneous and confused atmosphere. Aniline blue might be mistaken as specific for fungi (page 17); colloids not precipitated by electrolytes are said to be known as emulsoids, and the student could infer that semipermeability is related to non-passage of colloids (page 26); expansion is said to occur when gelatine passes into the sol condition, but not when bichromate passes into solution (page 27)—in both cases the material becomes distributed throughout a larger volume of space; the cones of *Pinus* are not different in "kind", but in age (page 85), and at the age stated for Figure 40, the micropyle would be tightly closed and the tip of the nucellus disintegrated.

With verbal and factual rehabilitation, the book in its present layout could be one to recommend. Unhappily, it cannot be recommended as it stands now.

Re-Education of the Injured Shoulder. By R. Barrie Brookes, F.C.S.P.; 1959. Edinburgh and London: E. and S. Livingstone, Limited. 8½" x 5½", pp. 20, with 59 illustrations. Price: 12s. 6d. (English).

THIS monograph is written by a physiotherapist, who describes the results of treatment of 779 patients with traumatic lesions of the shoulder joint. He has attempted to prove that movement instituted at the earliest possible moment under careful supervision and with the avoidance of pain, yields the best results, and it would appear that the cases quoted prove that this principle is correct.

The text is divided into chapters on anatomy and function followed by a description of specific injuries and treatment. The first two chapters attempt to summarize the literature available on the subject; but the author's lack of medical training is apparent in the confusion of thought and poor presentation of the facts. The bibliography is full and useful. The chapters on specific diseases lay great emphasis on diagnosis, especially in lesions of the rotator cuff, this would appear to be unnecessary in a book written for physiotherapists, who should be carrying out specific instructions under medical supervision after a surgical diagnosis has been established. The lengthy description of tears of the rotator cuff does alert one to the fact that the injury is more common in patients over the age of 50 years than is usually recognized, and should receive more attention.

The details of physical therapy are concise and the illustrations clear, but the result does not give much information that is not already contained in the recognized textbooks.

Text-Book of Surgery. By Patrick Kiely, B.Sc., M.D., M.Ch. (N.U.I.), F.R.C.S. (England); Second Edition; 1958. London: H. K. Lewis and Company, Limited. 9¾" x 5¾", pp. 1168, with 605 illustrations. Price: £3 3s. (English).

PATRICK KIELY is the Professor of Surgery in Cork. Doubtless one can still count on seeing there the jaunting car, not hiding away in the back streets, but holding its own with the automobile on the crown of the road. There is in his writing, similarly, evidence of a dogged conservatism, of which he is no way ashamed. The pattern which he follows is traditional—one is tempted to say, old-fashioned. Here in the early chapters come gangrene, dry and wet, and ergotism and cancrum oris and noma, phagedænic ulceration of "prisons, jails and camps", and radium and even a few lines on atomic burns. ("Atomic energy kills three times—blast, heat and irradiation.") We can guess what will come next—septicæmia, pyæmia and erysipelas and the old favourites (now quickly being forgotten), syphilis with its corona veneris and Colles's law, and gonorrhœa with its Cowperitis and the painful heel.

We should never seriously consider buying this textbook, although the price is modest by modern standards. It is, however, the work of a single author, and it comes as no surprise when we read in the preface that he has " . . . put his lectures into book form . . . ". It has that cohesion and completeness which are often missing in the multiple author works to which we have long since become accustomed. Kiely holds to the view that, in the undergraduate teaching of surgery " . . . the whole subject must be presented in a general way and must be reasonably balanced". We find ourselves in complete harmony

with this philosophy, and we should like to believe that therein lies the merit of the book—for merit it must have, since we are now welcoming a new edition. Any young man who believes he could do better with this approach (and many could) would do well to follow Kiely's example.

A Symposium on the Evaluation of Drug Toxicity. Editors for Imperial Chemical Industries, Limited: A. L. Walpole, Ph.D., B.Sc., and A. Spinks, M.A., Ph.D., B.Sc.; 1958. London: J. and A. Churchill, Limited. 9¼" × 5¾", pp. 152, with 58 illustrations. Price: 25s. (English).

THIS book is an account of a symposium held in 1957 to mark the opening of the new research laboratories of Imperial Chemical Industries at Alderley Park in England. It comprises, therefore, a number of separate papers from distinguished scientists specializing in various aspects of pharmacology and therapeutics; many manifestations of drug toxicity are considered.

The first contribution, by Professor E. J. Wayne, is concerned with the problems of toxicity in clinical medicine; but the major part of the book is devoted to discussions on the assessment of toxicity by animal experimentation. An interesting paper entitled "The Determination of Efficacy and Safety of New Drugs" by Dr. J. T. Litchfield, of the American Cyanamid Company, discusses the general principles involved in searching for and elevating interesting new drugs. Testing of drugs for toxicity, allergic reactions, hazards in the use of new drugs, and the toxic action of drugs on the bone marrow and on the liver were all subjects for discussion. At the conclusion of each main chapter two or three pages of discussion are given. The participants in the symposium have in this way amplified and further explored points raised in each paper.

This book will be of considerable interest to the pharmacologist; but its value is greater than this alone, as it provides a fascinating and readable background to what is today a major industry, the search for new therapeutic substances for human and veterinary medicine. It is the sort of book which the keen practitioner, who is anxious to extend his field of interest beyond his day-to-day work, should read. Each speaker introduces his subject very clearly, and the scope of the book does not require a detailed knowledge of medical science for its understanding or appreciation.

It is to be hoped, as is suggested in the foreword, that this symposium will be followed by others. Surely today the time has come when we must pause and assess the problems of industrial pharmacological development in this way.

Emergency War Surgery. U.S. Armed Forces Issue of Nato Handbook, Prepared for Use by the Medical Services of Nato Nation. United States Department of Defense; 1958. Washington: United States Government Printing Office. 7¾" × 5", pp. 424, with seven tables and 17 illustrations. Price not stated.

THE NATO handbook "Emergency War Surgery" is an attempt to give a highly compacted and crystallized review of the present opinions on the complex subject of war surgery. It was developed by a committee of surgical consultants from France, the United Kingdom and the United States, who have no doubt had access to all the best and most modern material available. The task must have seemed a most formidable one. To cover virtually all branches of traumatic surgery in an authoritative way, to deal with the myriad problems involved and to condense the result into a small handbook which could be carried fitted to the pocket of the field uniform is no mean task.

The result is a highly distilled essence, which has achieved a high degree of success. The entire field of traumatic surgery has been covered, and whilst much of it is in brief, all the major items are dealt with. Clear, lucid and simple expositions are given of the basic physiopathological processes concerned with most types of wounds which can be inflicted by the terrible weapons of destruction of modern warfare. The management of the individual casualty is outlined for both single and multiple injuries of many sorts, and the instructions given are applicable to the solitary casualty, or to one of the mass of casualties resulting from the explosion of a nuclear weapon. Clearly, many of the problems that arise in the event of such a disaster are essentially insoluble; but the authors have not avoided this issue, and have done their best to offer pertinent comment on those features which, under these circumstances, would most require consideration. In many, situations such as this would engender a feeling of dismay and despair; to those so affected this small publication should provide both comfort and help, as it has a useful and effective approach.

Any greatly simplified expression of opinion of a big and complex problem is always open to much criticism. The critic can invariably find something that could have been better done or should have been differently presented. This publication, however, would seem to have achieved its objects, so that such carping comment is out of place. Any harsh critic should first try to achieve these ends himself.

No longer can service surgery be divorced from civilian surgery. Everyone is now at risk. For those who are properly aware of this fact, this publication will prove helpful. A very small proportion of the medical population are surgeons; many doctors will feel that under certain circumstances they could be found wanting and inadequate. For these, and for the organized civil defence units, this small volume will be useful.

Progressive Exercise Therapy in Rehabilitation and Physical Education. By John H. C. Colson, F.C.S.P., M.S.R.G., M.A.O.T., with a foreword by J. M. P. Clark, M.B.E., M.B., Ch.B., F.R.C.S.; 1958, Bristol: John Wright and Sons, Limited. 8½" × 5½", pp. 196, with 174 illustrations. Price: 18s. 6d. (English).

IN rehabilitation, active therapy in the form of exercise is of paramount importance. In this book, Mr. Colson, well known for his work in remedial gymnastics, discusses exercise therapy. Paying attention to static exercises and to exercises for increasing range and power, he deals in turn with various regions of the body, and the text is illustrated with very clear drawings. He briefly discusses exercise therapy in certain surgical conditions, and concludes with a chapter devoted to general exercises by the use of circuit training and exercises to music. The most valuable aspect of the book, however, is the careful attention which has been devoted to progression in exercise. This is often the most difficult part of exercise therapy, and yet it is most important.

This book has been designed, not as a textbook, but as a handy reference guide to all those using exercise therapy, and its very compactness and clarity of illustration enhance its value in this regard. It should prove invaluable to physiotherapists and remedial gymnasts, and to all those interested in the field of rehabilitation.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Chest and Heart Disease in the Commonwealth"; 1958. London: National Association for the Prevention of Tuberculosis. 8½" × 5½", pp. 448, with illustrations. (Price not stated).

The full transactions of the N.A.P.T. Commonwealth Chest Conference of the British Tuberculosis Association, 1958.

"Nobody Need Be Fat", by R. Kemp, T.D., M.D., M.R.C.P.; 1959. London: William Heinemann Medical Books Limited. 7¼" × 4¼", pp. 155. Price: 12s. 6d. (English).

A "popular" book on the dietetic approach to slimming.

"Early Diagnosis", edited by Henry Miller, M.D., F.R.C.P. First edition; 1959. London and Edinburgh: E. & S. Livingstone. 8½" × 5½", pp. 408. Price 25s. (English).

A book for the general practitioner.

"Shakespeare and Medicine", by R. R. Simpson, M.B., Ch.B., F.R.C.S., F.R.C.S.Ed., 1959. Edinburgh and London: E. & S. Livingstone, Limited. 8½" × 5½", pp. 170. Price: 25s.

A detailed examination of the medical references in Shakespeare's plays and poems.

"Tetanus Neonatorum", by B. A. S. Gerungan, Med. Drs. Arts., Djakarta: Dedicated to World Health Organization. 7¾" × 5¼", pp. 16. Price: not stated.

Some experiences with the serious problem of tetanus neonatorum in the Celebes.

The Medical Journal of Australia

SATURDAY, AUGUST 29, 1959.

INTERNATIONAL ERADICATION AND CONTROL OF INFECTIOUS DISEASES.

ALTHOUGH it may provoke a doubtful smile here and there, the World Health Organization's latest approach to certain infectious diseases, that of eradication, certainly stirs the imagination. To us in Australia, of course, many of the most devastating infectious diseases seem quite remote. We have not been seriously troubled, when at home, by smallpox, cholera, plague, yellow fever or even, to any significant extent, malaria—and there has been no recorded outbreak of the last-mentioned disease on the mainland for the past decade. For this freedom we must largely thank our quarantine laws and the isolated geographical position which facilitates their administration. We cannot regard ourselves as exempt from all these diseases, despite our high standard of hygiene and general living conditions, and modern speed of transport has increased the hazard. Consequently, even in Australia, quite apart from any concern which we should have for the welfare of mankind as a whole, we might well be pleased if a major infectious disease just disappeared from the face of the earth.

This is in fact what WHO is planning to do with malaria. The policy of eradication was adopted in the first place largely because of the development of resistance to chlorinated hydrocarbon insecticides by vector mosquitoes, and a degree of urgency exists because of the difficulty of knowing what further resistance may develop. According to the Annual Report of the Director-General of WHO for 1958, substantial progress has been made. In the Americas, with the exception of only one country, the conversion of national control projects into eradication programmes has been completed. In Africa, promising results achieved by the various pilot projects foreshadow the possibility of eradication in the very near future. In the Eastern Mediterranean region, six countries are now engaged in active eradication, while five others are planning the conversion stage. With India's adoption of eradication as the target for antimalarial work, the number of people now covered by eradication schemes has increased by more than 350,000,000. Malaria has disappeared from regions of the globe whose population, totalling 300,000,000, was previously exposed to the risk of the disease. In still other countries and territories, with 700,000,000 to 800,000,000 inhabitants, eradication campaigns are being either implemented or prepared. Thus of 1,300,000,000 people who, only a few years ago, were actual or potential victims of malaria, only 250,000,000 still remain unprotected by eradication measures. The report does not, however, make light of the difficulties. Eradication, it is agreed, is a very complex and difficult undertaking, and governments must realize their responsibilities for carrying it through to a successful conclusion. One may question the practicability of wholly eliminating the reservoir of infection among primitive peoples with as yet limited contact with the outside world, but the broad idea seems well worth a trial, and it is still of value to eradicate the disease over large areas at least.

The eradication of smallpox is perhaps a more hopeful proposition, for, as the Director-General of WHO, Dr. M. G. Candau, pointed out to the Twelfth World Health Assembly recently, we have known for more than a hundred and fifty years how to prevent smallpox, and this disease has, in fact, already been wiped out over vast regions of the world. However, as he emphasizes, if there is one disease in the face of which the world remains indivisible, it is smallpox; so long as it survives in a single country it constitutes a danger to all, and calls for costly and complicated precautionary measures. Until last year, for one reason or another, it had not been possible to establish the necessary plan of international action essential for the eradication of the disease, but a decision of the Eleventh World Health Assembly in 1958 set the machinary going which may well, as Dr. Candau has said, free the world of a burden which, in the light of our knowledge, is a complete anachronism. Perhaps also it is not too much to hope that yaws will respond completely to the vigorous attack now being made on it on a world-wide basis. To curb undue optimism the possibility has been raised of invasion by venereal syphilis of areas cleared of yaws, but WHO is aware of this threat.

Influenza and other respiratory virus diseases would undoubtedly constitute a much harder nut to crack; a great deal more would need to be known about them than is known at present if we were to hope to eradicate them. However, apart from the variable threat which they offer to life, they have a tremendous nuisance and economic significance which warrants the most strenuous efforts to overcome them. This will certainly never be accomplished other than on an international level, for these diseases know no frontiers and show little respect for even the most thorough quarantine systems. Fortunately this is not so for another dangerous virus disease, yellow fever, but probably only because international control measures are rigidly enforced. As was pointed out in a comment[1] on Sir John Boyd's 1958 Blackham Lecture,[2] reservoirs of infection persist in the jungles of West Africa and Central and South America, and since 1948 a wave of virulent yellow fever has been sweeping northwards from Panama through Central America, and in places practically annihilating the monkey population. It has now reached the forests of Mexico, where it may spontaneously end. Fortunately, few cases have occurred among men because of the complete protection offered by vaccination. Why the disease has not spread to other parts of Africa is at present something of a mystery, as is its absence from Asia, where suitable mosquito vectors are not lacking. Certainly no one would like to see it get loose in the heavily populated Asian countries, and every precaution is taken. A striking and, in retrospect, amusing example

[1] *The Times*, February 6, 1959.
[2] *J. roy. Inst. publ. Hlth*, 1959, 22: 11 (January).

of this fact was related earlier this year in a letter to *The Times* by Herbert Thompson.[1] It appears that Sir Stafford Cripps, when he flew to India in 1942 to try to shift the political deadlock caused by the opposition to the war on the part of the Indian National Congress, neglected to be inoculated against yellow fever. Before his arrival this omission was discovered by the Public Health Commissioner, who promptly forbade Sir Stafford's landing. Apart from anything else, this was a major political complication, which the Viceroy found personally very embarrassing. The writer to *The Times* goes on:

> In the end with the help of such medical authorities as he could gather the Viceroy persuaded the Public Health Commissioner to yield so far as to allow Sir Stafford to disembark at Karachi alongside what was virtually a fly-proof meatsafe, spend the night in it while further precautions were being taken, and then fly on to New Delhi. There he was bundled into an air-conditioned car and decanted into an air-conditioned room in Viceroy's House. In that he was immured until the period of incubation was over. Sir Stafford had to submit, though he had intended to conduct his talks well outside the periphery of the Viceregal circle.
>
> Later I asked the then Director General of the Indian Medical Service what he would do if someone like Sir Stafford imported yellow fever from Africa. "I would board the first aeroplane to Ceylon", was the grimly humorous answer.

Yellow fever is thus obviously not taken lightly by those responsible for international health control, and rightly so. Vaccination against it is effective, but its possible impact on a large susceptible population such as that of India is awesome to contemplate. We should not lightly forget that yellow fever, amongst other manifestations of its power, made de Lesseps abandon his attempt to build the Panama Canal. If the World Health Organization manages to confound the sceptics on its malaria eradication campaign, perhaps it will turn its attention to this other mosquito-borne havoc-maker.

Current Comment.

TRYPSIN IN INFLAMMATORY CONDITIONS.

TRYPSIN, introduced parenterally, has been used spasmodically for over fifty years as an agent in dissolving inflammatory tissue and thrombo-embolic formations. During the past few years there has been a renewed interest in its use, and three papers have recently appeared claiming good results in the treatment of very different conditions. T. G. Glass and A. Fischer[2] have used parenterally introduced trypsin in crystalline form in the treatment of œdema following trauma, H. Murphy[3] has used it in the treatment of 210 patients with peripheral vascular diseases, and R. Neubauer, J. Esposito and A. Sindoni[4] have used it to treat two diabetic patients with the nephrotic syndrome.

Glass and Fischer treated 20 children suffering from washing machine wringer injuries of the arm with intramuscular injections of lyophilized crystalline trypsin in 5% gelatin solution. A similar group of 20 children were not given trypsin. There was considerable œdema in the injured arm, and this could be measured by determining the circumference of the injured arm at three different levels daily during treatment and comparing this with the measurement at the time of the patient's admission to hospital. On treatment with trypsin only occasionally did the circumference increase, and that only on the first day. Of the patients not treated with trypsin half showed an increase, which lasted up to five days. The tenderness in the arm diminished much more rapidly in the trypsin-treated patients. Glass and Fischer consider that the results of their study indicate that trypsin given parenterally is beneficial in the treatment of soft tissue œdema and is safe in children.

A survey of the literature of the past seven years shows that extensive use has been made of typsin in peripheral vascular disease with varying results. H. Murphy has made a study of 210 patients with peripheral vascular disease treated in office practice with intramuscular injections of crystalline trypsin in 5% gelatin solution. The conditions treated include varicose ulcers; of 42 patients treated the results were classified as excellent in 41 and good in one. "Excellent" means complete healing of the ulcers. Of 33 patients with superficial venous thrombosis, all showed complete dissolution of the thrombus and recanalization of the vein or veins. Of 129 patients with some form of thrombophlebitis, 81 showed excellent results, 42 good results and six poor results. Of the whole series results were excellent in 75% and good in 22% of the patients. Two patients showed generalized urticarial reactions.

Neubauer, Esposito and Sindoni used crystalline trypsin in the treatment of quite different conditions and in quite a different manner. They showed first that trypsin could be absorbed through the membranes of the mouth, and that 30 mg. could be absorbed daily with no untoward reactions. By this method they treated two patients with diabetes and the nephrotic syndrome. The two patients showed considerable clinical and laboratory improvement with the buccal administration of trypsin in large doses over a prolonged period. In particular, the œdema disappeared and has not recurred, and the patients are both completely asymptomatic and working full-time.

The mechanism by which parenterally introduced trypsin works has not been demonstrated, although a number of theses have been put forward, none of which seems to be likely. These papers and papers published in the past few years seem to make it clear that parenterally introduced trypsin can be of great use in the management of many kinds of inflammatory and œdematous changes in the body.

[1] *The Times*, February 18, 1959.
[2] *A.M.A.J. Dis. Child.*, 1959, 97:571 (May).
[3] *N.Y. St. J. Med.*, 1959, 59:1973 (May).
[4] *Amer. J. med. Sci.*, 1959, 237:559 (May).

SKIING INJURIES.

"SKIING is here to stay. Its ever-increasing popularity can be witnessed by the number of ski lodges and resorts that have sprung up in all parts of the United States"—and, one could well add, "of the Australian Alps". With skiing comes the problem of the "ski injury", which is the subject of a paper by Hans Kraus.[1] In this he discusses the problem of prevention of skiing injuries and outlines the principles of treatment by the technique of early mobilization assisted by surface anæsthesia. *Questionnaires* were sent out to a number of physicians in skiing resorts, to ski patrol men, to ski schools and to prominent skiers in an endeavour to obtain more information on the cause and prevention of skiing injuries. It appears that poor muscular condition is a main cause of injury; physical conditioning before skiing is the best prevention. A Westchester, N.Y., ski club averaged 5000 skiing days in 11 years. During this time pre-ski conditioning was practised. No fractures, and only two mild sprains, occurred. When the pre-ski conditioning was given up, three fractures occurred during the first year. Equipment, too, is important. While release bindings are by no means safety bindings, they do offer a worthwhile improvement on the chance of avoiding injury. At Garmisch-Partenkirchen, Germany, of 17,500 persons who rented non-release bindings, 74 sustained fractures; of 45,043 who rented release bindings, 11 sustained fractures.

[1] *J. Amer. med. Ass.*, 1959, 13:1414 (March 28).

When injuries have occurred, their management depends on generally accepted orthopædic principles; in an endeavour to restore full function in the shortest possible time, Kraus advises immediate mobilization of many injuries. Fractures treated by this method include fractures of the pelvis (os pubis), fractures without displacement of the short bones of the foot (intraboot injury), fractures of the head of the humerus, fractures of the metacarpals, fractures of vertebræ, if uncomplicated and if no pedicles are involved, fractures of transverse processes of vertebræ, and fractures of the ankle without displacement of the lateral malleolus, if not combined with ligament tear. He prefers immediate mobilization for reduced dislocations of the shoulder and elbow, and for fractures of the tuberosity of the humerus. Immediate mobilization of sprains of the ankle and knee and strains of the lower part of the back, of the calf, or of the hamstring tendons is recommended. However, in ligamentous injuries it is considered important to exclude major ligament tears, which frequently need surgical repair or immobilization in plaster casts. The treatment is carried out in the following way. The pain is located by active movement, and ethyl chloride is sprayed on the area concerned. The patient then starts active movement of the part involved, in the direction in which movement has been painful and limited. As the patient increases his range, new painful areas will develop. These areas must again be sprayed and movement continued. Treatments last from 10 to 30 minutes, and should be carried out within limits of pain. The exercises should be performed from a minimum of twice a day to as often as once every hour, for approximately five minutes. There is no adequate scientific explanation for the deep effect of surface anæsthesia. Kraus claims that the use of surface anæsthesia, in preference to the injection of procaine hydrochloride into the muscles and ligaments, has definite advantages. However, he emphasizes the fact that exercise is an indispensable part of the treatment and that surface anæsthesia alone, without active movement, will not achieve good results. Many surgeons may prefer to follow established methods of treatment in which a preliminary period of immobilization, for days or weeks, precedes active exercise of an injured limb. However, Kraus has found the technique of immediate mobilization most effective in over 25 years' work with this method.

MACROGLOBULINS AND MACROGLOBULINÆMIA.

MACROGLOBULINÆMIA is an entity which was first described just over ten years ago, and which has recently received considerable attention. Its study demands the use of the most modern serological and immunological techniques, no doubt one reason why it had previously escaped detection as a distinctive clinical and pathological condition. I. R. Mackay, of the Walter and Eliza Hall Institute of Medical Research, Melbourne, has recently contributed a review[1] of this condition, in which he describes the more important clinical manifestations and relevant biochemical and pathological aspects of macroglobulinæmia, with an appraisal of present concepts of this disease process, and gives details of 10 personally investigated cases. He states that macroglobulins are serum globulins of high molecular weight, which are detectable by ultracentrifugal analysis of serum. They normally comprise up to 4% of the total serum proteins, and include such substances as properdin, the Wassermann antibody and the rheumatoid factor. A minor increase in the serum concentration of macroglobulins may occur in nephrosis, cirrhosis, lupus erythematosus and congenital syphilis; however, the term macroglobulinæmia is by convention used only to describe a condition characterized by a gross and autonomous overproduction of an anomalous macroglobulin component of the serum, and usually associated with morbid effects. The main features of the condition are described by Mackay as follows:

> The disease occurs between the ages of 50 and 70 years, and seems to be more common in males . . . The major symptoms are weakness, lassitude, a bleeding diathesis, recurrent infections and disturbances of vision. The signs are those of anæmia, occasionally mild œdema, lymph-node enlargement, hepatosplenomegaly and eye-ground changes. These clinical features are determined by two factors: (*a*) systematized hyperplasia of cells of lymphocytic origin, causing marrow failure and enlargement of the liver, spleen and lymph nodes; (*b*) the physical presence of abnormal globulins in the blood, which may damage vascular endothelium and the surface of circulating cells, adsorb clotting factors, and cause "sludging" of blood in retinal capillaries and cold-sensitivity phenomena.

Other clinical manifestations of the condition may be extremely varied. Raynaud's phenomenon and cold sensitivity may occur if the macroglobulins behave like cryoproteins and precipitate on cooling. Ocular changes are important and may be the presenting symptom of the condition. Bizarre neurological changes have been reported, and may be associated with psychiatric changes; these manifestations are probably due to infiltrations of lymphoid cells and protein exudates in the central nervous system. Osteoporosis may occur, but focal bone changes are rare, and this is important in distinguishing the condition from multiple myelomatosis. Bence Jones proteinuria is not infrequent, but macroglobulins do not appear in significant concentration in the urine, and renal lesions are rare. Macroglobulinæmia has been reported in association with cancers of several kinds, but this association may be coincidental.

Mackay states that the diagnosis of macroglobulinæmia should be considered when the physician is confronted by an elderly patient with atypical reticulosis, lymphoma or lymphatic leukæmia, especially if mucosal bleeding is a prominent symptom. Filter-paper electrophoresis will practically exclude any condition other than multiple myelomatosis; differentiation from the latter condition is usually possible because of the absence of focal lesions, but may depend on ultracentrifugal analysis of serum.

The prognosis is variable. In most cases of the disease the outcome is eventually fatal, the patient dying after a long illness characterized by wasting, intercurrent infections and sepsis, bleeding and, rarely, cardiac insufficiency. Not infrequently death is precipitated by a cerebral hæmorrhage. In a few cases the disease runs a rapid course, and in others the patient may survive for many years without serious disability, ultimately dying from some unrelated cause. Apart from blood transfusions to correct anæmia, treatment is mainly supportive. Corticosteroids, cytotoxic agents, deep X-ray therapy and radioactive phosphorus have all been tried, with varied and uncertain results.

The pathological basis of the condition leaves room for interesting speculation. It has been suggested that macroglobulinæmia is an auto-immune phenomenon, but Mackay favours the idea that it is due to a pathological modification in the reticulo-endothelial system, belonging to that obscure borderland on the fringe of such frankly malignant conditions as lymphoma and myeloma. He presumes that this modification is a somatic mutation, the descendants of the mutant cell proliferating throughout the reticulo-endothelial system. It is suggested that such modified cells may retain some functional activity and synthesize a variety of pathological globulins, whose presence is manifested clinically by such conditions as macroglobulinæmia and cryoglobulinæmia. Macroglobulins may exhibit the properties of antibodies, particularly autoantibodies, but Mackay considers that "macroglobulin synthesis is essentially an autonomous rather than a reactive process, and the occasional presence of immunologically reactive sites on macroglobulin molecules must be regarded as an incidental phenomenon". Mackay concludes with a plea for a greater awareness of macroglobulinæmia, because the recognition of additional cases must lead to a better understanding of a condition which has an important bearing on problems concerned with antibody production, somatic mutation and cancer.

[1] *Aust. Ann. Med.*, 1958, 8:158 (May).

Abstracts from Medical Literature.

OBSTETRICS AND GYNÆCOLOGY.

Pregnancy in the Patient with Ileostomy and Colectomy.

F. O. Priest, R. K. Gilchrist and J. S. Long (*J. Amer. med. Ass.*, January 17, 1959) discuss the problem of pregnancy and delivery after ileostomy and colectomy. Seven such patients, who had 13 pregnancies, resulting in 10 living children, three early abortions and no maternal deaths, are briefly reported. Observations are made on these patients and on 22 similar pregnancies reported in the literature. It is generally agreed that ulcerative colitis has little effect on pregnancy, but pregnancy may bring about adverse and unpredictable effects on patients with ulcerative colitis. The problem is being encountered more frequently since ileostomy or ileostomy and colectomy are in common usage for uncontrolled and complicated cases of ulcerative colitis. Among the 10 pregnancies at term in the series quoted, nine patients were delivered per vaginam and one by Cæsarean section. Three patients had evidence of partial small bowel obstruction; all responded to medical treatment. One patient developed an intussusception at six months' gestation and required a revision of the ileostomy, and later a resection of the terminal ileum after delivery by Cæsarean section. The authors conclude that: (i) the presence of an ileostomy does not make subsequent pregnancy more difficult; (ii) young women with intractable colitis can be treated by ileostomy and colectomy without fear of subsequent inability to bear children, and can expect a normal delivery in most cases; (iii) adequate ante-natal care implies teamwork by the surgeon, the gastroenterologist and the obstetrician.

Fœtal Distress.

J. Walker (*Amer. J. Obstet. Gynec.*, January, 1959) states that, normally, in late pregnancy the oxygen levels in the blood of the fœtus are moderately high. Certain abnormal conditions lower this, namely: (i) rising gestational age, especially in primigravidæ; (ii) previous threatened abortion; (iii) maternal pre-eclampsia; (iv) prolonged or mechanically difficult labour; (v) anæsthesia. A slowing of the fœtal heart rate is generally considered a relatively dangerous sign, indicating the need for interference, and very few babies are lost in such cases. The authors consider, however, that we have been unwilling to accept meconium staining and fœtal heart rapidity as danger signs, and that more babies are lost in these circumstances than should be the case. He considers meconium staining as a sign of interference with the transplacental transfer of oxygen. Cæsarean section is not indicated on this sign alone, except when it is seen before the beginning of labour or early in labour in the case of a primigravida over 30 years of age. Meconium staining sometimes follows the use of "Pitocin", quinine or castor oil in medical inductions. When meconium first appears in a prolonged labour, for example one that has lasted over 24 hours, the mother should be given oxygen, and the case assessed. If labour is likely to last many more hours, delivery by Cæsarean section is strongly indicated. When associated with accidental hæmorrhage and pre-eclampsia, meconium staining is a particularly dangerous sign. Rapidity of the fœtal heart is seen in labour in the presence of infection, and often when the membranes have been ruptured for many days, even in the absence of maternal pyrexia. It may also be seen when labour is prolonged, and if associated with meconium staining the need for delivery appears to be urgent. It is impossible to ignore the strong possibility that many of the babies who survive in spite of such signs of distress are permanently damaged.

Excessive Fœtal Development.

R. N. Bolton (*Amer. J. Obstet. Gynec.*, January, 1959) presents a study of 144 infants who weighed 4500 grammes or over at birth. He states that there is no specific aspect of the pregnant patient which is pathognomonic of excessive fœtal development, but that certain factors point to it. These are: (i) a large-statured multipara with a past history of babies weighing 4500 grammes or more; (ii) a maternal weight gain in excess of 24 pounds over ideal non-pregnant weight; (iii) the prolongation of pregnancy beyond 293 days after the last menstrual period; (iv) poor progress of labour in a patient with a good obstetrical history; (v) a palpably large fœtus. Few women who are delivered of large infants will have diabetes at the time, but an appreciable number will develop diabetes later on. Labour is not usually prolonged, and abnormal presentations are not common. However, severe injury to the fœtus is more frequent with excessive development, and Cæsarean section seems preferable to a difficult vaginal delivery. Post-partum hæmorrhage constitutes a real maternal hazard, and in all cases this must be anticipated.

A New Preparation for the Prevention of Lactation.

J. B. Watrous, R. E. Ahearn and M. A. Carvalho (*J. Amer. med. Assoc.*, January 17, 1959) report investigations to evaluate the efficacy and possible side effects of "Deladumone" as an inhibitor of lactation. This is a long-acting androgen-œstrogen combination which contains 90 mg. of testosterone œnanthate and 4 mg. of œstradiol valerate per millimetre in sesame oil. A single dose of two to four millilitres was injected parenterally at times ranging from the end of the first stage of labour to eight hours after delivery. Among 310 women who did not wish to breast feed their infants, 185 had a single injection of "Deladumone", 83 patients received an oral androgen-œstrogen preparation thrice daily during their six days in hospital, and 42 patients had no hormone medication. Results were assessed by the degree of post-partum engorgement of the breasts, breast discomfort and the necessity for analgesics and support of the breasts. The incidence of engorgement among 268 hormone-treated patients was 8·3%, as compared with 69% among 42 patients not so treated. The parenteral medication ("Deladumone"), when injected in doses of 3 or 4 ml., was more effective than oral medication ("Tylosterone"). A study of dosage and timing of the injection led the authors to conclude that the most effective parenteral injection was a single one of at least 3 ml., given at the end of the first stage of labour or within the first hour after delivery. They consider the preparation "Deladumone", when given in this way, to be safe, convenient and effective, and free from any undesirable withdrawal reaction.

B. Lo Presto and E. Y. Capinar (*J. Amer. med. Ass.*, January 17, 1959) report the results of a trial of "Deladumone" on 197 women with a view to preventing the onset of lactation. The patients were given single injections of 2 or 3 ml. at various times from between the first and second stages of labour to within two hours after delivery. The most effective method was found to be a dose of 3 ml., given after the first stage of labour. This completely suppressed breast symptoms in 43 out of 47 patients without affecting the course of labour, uterine involution or other physiological adjustments. Inhibition of lactation by this combined hormone was overcome in two instances when mothers later decided to breast feed their babies. No relationship could be established between the response to therapy and the age, parity or previous nursing experience of the patients. The authors conclude that post-partum lactation and its associated breast manifestations can be prevented in a high percentage of mothers by the administration of a single injection of at least 3 ml. of "Deladumone" at the end of the first stage of labour.

Surgical Management of the Tubal Factor in Infertility.

R. W. Cauwet (*J. Amer. med. Ass.*, October 25, 1958) surveys the results of operation on 69 patients with blocked Fallopian tubes during the five years ending June, 1957. The diagnosis of tubal occlusion was made by repeated Rubin's tests, salpingograms and culdoscopy, which was performed if there was doubt about blockage shown by salpingograms, if there was suspicion of endometriosis or pelvic inflammatory disease, or if infertility was still unexplained. Four standard operations were employed, namely salpingolysis, salpingostomy at the fimbria, resection of the blocked part with reanastomosis, and reimplantation of the tube into the uterus. In all procedures other than salpingolysis the use of polyethylene tubing as a splint to be left in place for as long as three months was an important part of the technique. Eighteen of the patients became pregnant, resulting in 13 live births; two pregnancies resulted in ectopic gestation and three in abortions. Of the 35 patients who had a salpingolysis, 14 became pregnant, resulting in 11 living children, two abortions and one ectopic pregnancy. In 14 cases salpingostomy at the fimbrial end was performed; in nine of these patency was demonstrated, but no pregnancy resulted. In 20 cases resection of the blocked area or tubal

implantation was performed; in nine of these patent tubes were demonstrated, and in four cases the patient became pregnant. The author stressed that, in spite of patency, pregnancy seldom occurs when the tubal wall has been badly damaged by disease. Pregnancy after surgery will result most frequently when damage is limited to peritubal adhesions. Culdoscopy may be necessary for the diagnosis of the adhesions.

Anæmia in Obstetrics and Gynæcology.

J. A. PRITCHARD (*Amer. J. Obstet. Gynec.*, January, 1959) surveys the use in obstetrics and gynæcology of parenteral iron for patients suffering from anæmia. The leading causes of anæmia were acute blood loss, chronic blood loss, frequent pregnancies with iron depletion, or some combination of these. Obstetrical patients who received supplementary iron during the ante-partum period came to delivery with a higher hæmoglobin level than did patients who had received no iron. In pregnancy, iron-dextran, given intramuscularly, did not improve the blood count to a significantly greater degree than when ferrous gluconate was given orally. The intramuscular administration of iron, however, seemed of considerable value for treating the severely iron-deficient patient to ensure maximum response late in pregnancy, and subsequently to correct promptly and completely depleted iron stores. The intramuscular injection produced only transient local soreness, whereas the use of saccharated iron oxide given intravenously was frequently associated with distressing reactions.

The Cytological Diagnosis of Genital Cancer.

W. J. ESTRADA *et alii* (*Amer. J. Obstet. Gynec.*, January, 1959) present an evaluation of the results obtained by the use of cytology in the detection of malignant conditions of the cervix from the study of 25,000 consecutive smears from 17,000 women. Both pre-malignant and borderline lesions are included under malignant conditions of the cervix in this work. As regards the site of collection, 60% of the smears were cervical and 32% were vaginal, the former yielding 77% of the suspicious or "positive" smears and the latter 19%. It is considered that the cervix yields a greater concentration of malignant cells and a better quality smear without cellular distortion. The results were as follows: 262 smears (1·05%) were classified as suspicious or "positive"; "false positive" smears constituted 0·14% of the total series, and 13% of the total number of the cases classed as suspicious or positive; there were four "false negative" smears in cases in which biopsy of the cervix had revealed carcinoma-in-situ; there were seven "false negative" smears in cases of endometrial carcinoma (21 cases of carcinoma of the endometrium were found, in four of which the diagnosis was made by examination of an endometrial aspiration smear); 148 previously unsuspected neoplasms of the cervix were discovered, in over 60 of which cases the appearance of the cervix was recorded as being essentially normal (eight of these apparently normal cervices were found to be the seat of invasive carcinoma). Out of 250 smears taken during pregnancy or the puerperium, five were classed as suspicious or malignant, but two of these were "false positives", carcinoma-in-situ was present in two other cases, and invasive carcinoma in the fifth. The average age of patients with invasive carcinoma of the cervix was 50·5 years, and of those with carcinoma-in-situ 37·5 years. From women less than 30 years old there were 26 suspicious smears and six "positive" smears. The authors suggest that cervical or vaginal smears should be examined once a year for women under the age of 30 years, and every six to 12 months thereafter. The discovery of "positive" smears should be followed by biopsy. They recommend that for women with post-menopausal bleeding in whom endometrial carcinoma is suspected, curettage with histopathological examination is preferable to the examination of cervical or endometrial smears.

Thyroid Tumours of the Ovary.

P. BROCQ, C. ROUVILLOIS AND J. GAUCHEZ (*Presse méd.*, January 28, 1959) present the results of a study of the types, prognosis and diagnosis of thyroid tumours of the ovary. Their work is based on unpublished work done by several colleagues, and on published material. They have collected 233 cases from the literature. They state that the most common type is that in which the thyroid tissue is histologically normal. Of the 233 cases, in 108 there were inclusions associated with a complex teratoma of the ovary; in 56 there were simple inclusions within the wall of an ovarian cyst (simple teratoma); in 69 cases the tumour was a pure ovarian goitre containing nothing but thyroid tissue. The development of such a thyroid adenoma may be accompanied by signs of toxicity. Occasionally the thyroid tissue resembles that of hyperthyroidism. In nine cases taken from the literature, the removal of the thyroid tumour only effected a cure; but in three cases surgical removal did not relieve the toxic manifestations, because both the thyroid body and the ovarian tissue were producing abnormal secretions. (This was proved by post-operative tests with radioactive iodine.) In only eight of the 233 cases was there certain evidence of malignant change. The diagnosis was only rarely made before operation. The type of aberrant thyroid tissue can be determined by a test dose of radioactive iodine.

The Underdeveloped Secretory Endometrium.

R. W. NOYES (*Amer. J. Obstet. Gynec.*, May, 1959) examines the problem of the underdeveloped secretory endometrium, with particular reference to the incidence of the condition, the difficulties commonly encountered in its diagnosis and the methods and results of its treatment. Secretory underdevelopment of the endometrium should be diagnosed only after at least two biopsies of the surface layers of the fundal endometrium at about the middle of the secretory phase have been made and compared with both the basal temperature shift and the onset of succeeding menses as points of reference to the time and length of the secretory phase. There should be at least two days' retardation of the endometrial response before the diagnosis of underdeveloped secretory endometrium is made. Histological examination of the endometrium is much superior to present histochemical methods in revealing secretory deficiencies. The author considers that the significance of underdeveloped secretory endometrium is overrated, as the ova of several species of mammals are known to survive deficient environments in the oviduct or in the uterus when the endometrium is retarded. His studies showed that there is little evidence that rather large doses of hormones given between times of ovulation and implantation alter the balance of the reproductive system. Œstrogen therapy appears to retard the secretory response, chorionic gonadotrophin advances the response slightly, but progesterone has no effect on endometrial development. It is concluded that the endocrine treatment of infertile and aborting patients with this condition must still be considered to be empirical rather than rational therapy.

Varicose Veins in Pregnancy.

J. C. WEE AND H. B. HATCH (*Amer. J. Obstet. Gynec.*, May, 1959) report a study of certain varicosities in pregnancy which are shown clinically by warm tender angiectases, which become symptomatic with change of position, indicating that there is an arterio-venous fistula in these areas. Oxygen saturation studies indicate a significant increase in the percentage of arterial blood in these areas when compared with venous blood from the opposite leg or the upper extremities. Administration of stilbœstrol, 25 mg. a day, results in improvement of symptoms. The same symptoms occur premenstrually in women who have had warm varicosities for a long time, and these too are improved by giving stilbœstrol.

ANÆSTHESIA.

The Effect of Fluothane, Cyclopropane and Ether on Liver Function.

D. M. LITTLE, JNR., C. M. BARBOUR AND J. B. GIVEN (*Surg. Gynec. Obstet.*, December, 1958) have compared the effect of fluothane, cyclopropane and ether anæsthesia on the liver by means of liver function tests performed on 30 patients who had undergone major pelvic or perineal surgery. The tests were performed pre-operatively and on the second and seventh days after operation. Post-operative abnormalities of liver function were detected in five of the 10 patients who received fluothane anæsthesia, four of the 10 who received cyclopropane anæsthesia, and eight of the 10 who received ether anæsthesia. The abnormalities noted often occurred only in an isolated test, and were in some cases of questionable significance. The authors conclude that the effects of anæsthesia on the liver revealed by this study were innocuous, and suggest that this may have been due to the excellent general health of the patients concerned, to the site and nature of the surgical procedures, or to the care taken to prevent any degree of hypoxia or hypercapnia.

Congresses.

NORTH QUEENSLAND MEDICAL CONFERENCE.

THE Third North Queensland Medical Conference was held at Mackay from September 22 to 29, 1958, with Dr. C. E. Williams as Patron, Dr. P. W. Hopkins as President and Dr. I. H. Chenoweth as Honorary Secretary.

Inaugural Meeting.

The Inaugural Meeting was held in the Parish Hall, Gordon Street, at 8 p.m. on Monday, September 22, 1958. Dr. P. W. Hopkins was in the chair. The Conference was officially opened by his Worship, the Mayor of Mackay, Alderman J. Binnington. The inaugural address was delivered by Sir Alexander Murphy.

The Treatment of Common Dermatoses.

DR. I. O. STAHLE (Melbourne) read a paper on principles of treatment of common dermatoses.

The Eczemas and their Treatment.

DR. C. T. PETHERBRIDGE (Townsville) read a paper entitled "The Eczemas and their Treatment". He said that eczema was an inflammatory reaction of the skin affecting both the epidermis and the dermis. The eczema reaction was a pattern reaction resulting from many exogenous and endogenous causes. It was a complex of naked-eye and histological changes. It might occur by itself, or accompany or complicate some other form of dermatitis. It was often allergic, but might be non-allergic, and could be produced, for example, by a physical agent or by a primary irritant. Certain non-specific factors, either alone or in combination, could predispose to the development of the allergic eczema reaction; their action had been likened to that of a catalyst. An accurate ætiological classification of the eczemas was not possible; but for practical purposes they might be divided into three types—the atopic eczema reaction, the non-atopic eczema reaction and the mixed reaction. The first type included the so-called infantile eczema, and had to be regarded as distinct from all other types for classification purposes, because the shock tissue appeared to be the cutaneous vessels. The second type had six groups of causes; (i) primary irritants; (ii) physical agents; (iii) non-microbic contactants, not primary irritants; (iv) microbes; (v) parasites; (vi) endogenous factors—drugs, foods, nutritional factors, viruses, hormones, metabolic factors, nervous and emotional factors, etc. The third type of eczema reaction, the mixed reaction, was common.

Turning to the mechanism of the eczema reaction, Dr. Petherbridge said that it was specific and acquired, and was produced by one allergen only, or by allergens which were closely related immunochemically. The mechanism of certain types of endogenous eczema was unknown, but some of them were perhaps allergic, the allergen being another tissue. All efforts to demonstrate antibodies in eczema had failed, but that did not necessarily mean that they did not exist. In allergic contact eczema, the whole skin usually became simultaneously sensitized, even when the allergen was applied only to a small area. Allergens which produced the allergic eczema reaction were usually relatively simple chemical compounds. Linkage of the simple chemical compound to a protein seemed to be the first step in eczematous contact sensitization. After experimental work, Haxthausen concluded that eczematous allergy was not due to any particular acquired property of the epidermal cells, but to a factor supplied to the skin from within, and further, that eczematous allergy was a state in which the active principle was supplied to the skin steadily, but was at the same time subject to destruction. As a result of further experimental work, Haxthausen decided that the active principle reached the skin by way of the blood-stream, and that it was remotely possible that the active principle might reach the skin from the underlying connective tissue.

Dr. Petherbridge then said that consideration of dissemination of the eczema reaction was of great importance in the management of the eczemas. The fact that the whole skin was usually simultaneously sensitized in allergic eczema prepared the ground for dissemination, which was common. New lesions appeared on sites distant from the primary lesions, with no evidence that the causal agent had made contact with the new sites. The process could continue, apparently spontaneously. A number of reasons for the phenomenon had been suggested; in practice, dissemination frequently followed the topical application of an unsuitable medicament, or bacterial infection, or rubbing and scratching, or some combination of those causes. Discussing residual eczema, Dr. Petherbridge said that it could persist in the absence of further exposure to the original cause. Persistence of a contact eczema reaction might be due to cross-sensitization, or to the operation of the factors producing dissemination, or to the allergen's remaining in the reticulo-endothelial system, or to the possible ability of one exposure to an allergen to set up more or less permanent changes in the antibody-synthesizing mechanism.

Referring to the ætiological diagnosis of the eczemas, Dr. Petherbridge said that a complete diagnosis would not be made in each case; but every effort should be made to determine the ætiology, since effective treatment depended on knowledge of the cause. The morphological appearance was not a reliable pointer to the cause, since it might vary at different sites in the same patient, and in different patients although the cause was the same. The distribution of the lesions was untrustworthy, except in allergic contact eczema. The first step was to determine whether the reaction was atopic or non-atopic. The patient's personal and family history and a characteristic eruption might suffice to allow a provisional diagnosis of atopic eczema to be made. The diagnosis was reinforced by the demonstration of eosinophilia, by the production of a high proportion of immediate weal reactions to scratch or intracutaneous tests with standard common foods and inhalants, and by a normal or below normal number of positive reactions to a standard series of patch tests. Atopic subjects had a high tendency to develop "infantile eczema", and the same eczema reaction might occur in older atopic subjects. However, atopic subjects could develop non-atopic eczema. Non-atopic eczema reactions had to be differentiated serially. There was no reliable way of distinguishing between the macroscopic appearance of (i) a primary irritant contact eczema reaction and that of (ii) an allergic contact eczema reaction, but the microscopic appearances differed. In (i) the exudate consisted chiefly of polymorphonuclear leucocytes, whereas in (ii) it consisted of mononuclear cells, mainly lymphocytes. In (i) necrosis was seen mainly near the surface, with subsequent extension of the reaction deeper into the epidermis; intraepidermal intercellular œdema occurred, but vesicle formation was less common and might be absent. Important leads in diagnosis in allergic contact eczema were the chronological order of events, the site of the initial lesion, the general distribution of the lesions, and the results of skin tests.

Discussing the treatment of the eczemas, Dr. Petherbridge said that the first aim should be to reach an early ætiological diagnosis and remove the cause. The reaction might sometimes persist after the original cause had been removed, because of the operation of secondary factors; therefore in all cases a search should be made for secondary factors. In some forms of endogenous eczema and in atopic eczema it would not be possible to find the cause; however, factors known to have an influence in such cases should be sought, and if possible removed or avoided. The eczema reaction should always receive local treatment, the type of which would be determined by the severity of the reaction. The general principle was, the more severe the reaction, the milder the treatment. As an alternative to local treatment, hydrocortisone or prednisolone might be applied in cases of allergic contact eczema, atopic eczema and seborrhœic dermatitis. An appropriate antibiotic, preferably one that could not be given systemically, could be added if infection was present, and even in the absence of infection. It appeared that the antibiotic increased the antieczematous effect of the corticosteroid, and the corticosteroid increased the antibacterial effect of the antibiotic. The best results were obtained by becoming familiar with the effects of a limited range of substances and vehicles, rather than by using a large range. The systemic use of ACTH or a corticosteroid, in addition to topical treatment, was warranted in carefully chosen cases of "infantile eczema", when the eruption was severe and extensive and the general health was deteriorating. Those measures were also warranted in certain cases of disseminated and residual eczema not responding to local treatment. However, it had to be remembered that ACTH and corticosteroids were purely suppressive agents. Superficial X radiation could be employed as an auxiliary measure with great advantage in the chronic stages of many forms of eczema.

Otorrhœa and Common Diseases of the External Ear.

DR. R. PALMERSTON RUNDLE (Rockhampton) read a paper entitled "Methods of Treating Otorrhœa and some Common Diseases of the External Ear".

The "Red Eye".

DR. L. LOWTH (Townsville) read a paper entitled "The 'Red Eye'". He said that there were three major causes

of the condition, as well as many lesser causes. The three major causes were glaucoma, iritis or iridocyclitis and conjunctivitis. Discussing glaucoma, Dr. Lowth said that there were two types, primary or simple, and secondary. Often its onset was insidious, and it was not recognized until irreversible damage had been done to the eye. The acute or congestive phase was a cause of "red eye". In that case the patient's chief complaint was pain, intractable and distributed over the whole of the fifth nerve area, often accompanied by nausea, abdominal pain and vomiting. The eye was suffused and lachrymating, the lids were often œdematous and the vision was much diminished. The cornea was hazy; the pupil was fixed and oval, semi-dilated with the long axis vertical, and if the eye could be palpated, the tension was much raised. Dr. Lowth said that such a situation was perhaps the most important ophthalmic emergency, because if treatment was not instituted rapidly and adequately, blindness would result in less than 24 hours. He then briefly discussed the ætiology of glaucoma, and went on to the second major cause of "red eye", iritis or iridocyclitis. He said that in most cases the ætiology was obscure, although it might be obvious, and severe infections had distinguishing clinical features. Generally all investigations gave negative results, and it was probable that the "red eye" was then not due to direct infection, but was rather allergic or metabolic. Causes might be classified in five groups—exogenous, secondary, granulomatous, allergic or metabolic. The redness was most marked near the limbus. As the iris was anatomically similar to erectile tissue, its general pattern became muddy, and its colour was slightly altered in comparison with the other eye. The pupil was constricted, owing to hyperæmia, and its edge was often irregular owing to adhesions from posterior synechiæ. Its reactions were sluggish. The eye was watery, and unless conjunctivitis was also present, the discharge was lachrymal. Pain was neuralgic in nature, but usually distributed only in the first division of the fifth nerve; it was more severe at night. Vision was diminished, but not so much as in glaucoma, and the intraocular tension might be raised as in secondary glaucoma. The urgency was not so great as in glaucoma. The diagnosis had to be certain, since the treatments for the two conditions were diametrically opposed to each other—miotics in glaucoma, mydriatics in iritis; the wrong use of either could be very damaging. The most difficult problem was iritis complicated by secondary glaucoma. In that case the cornea was likely to be œdematous, and one could not see enough to make a diagnosis. If there was any doubt, the instillation of two or three drops of pure glycerine into the eye together with hot bathing would often clear the cornea enough to allow one to see the iris and pupil and make a diagnosis, particularly if posterior synechiæ were seen. Hot spoon bathing in all cases was good first aid treatment. The administration of "Diamox" in a dosage of 500 mg. at once and 250 mg. every six hours was also of value in both primary and secondary glaucoma; "Diamox" should be exhibited if there was doubt as to the diagnosis.

Dr. Lowth then said that in the last of the three major causes, conjunctivitis, the clinical picture was well known. The condition was usually bilateral; it was often not so severe in one eye as in the other. Unilateral conjunctivitis should suggest exclusion of corneal foreign body, corneal ulcer, ingrowing eyelashes or dacryocystitis of the same side. It was often difficult to pick a transparent foreign body, such as the husk of a seed or the wing case of an insect, which if not removed early would slowly track across the cornea, leaving a permanent nebula. The ætiological agent was any one of the common pathogens or a combination of them. A well-known type was ophthalmia neonatorum, a notifiable disease, of which before the second World War only 17% was gonococcal; efficient ante-natal care had lowered the figure still further. The most common organism at present was the *Staphylococcus aureus*, whose resistance to penicillin was paralleled by its resistance to the more recent antibiotics such as chloramphenicol and the tetracycline group. Other less common causes of conjunctivitis were allergy, trauma and irritant gases and solutions; actinic conjunctivitis due to the electric welding flash was frequently seen. Trachoma was a special type of conjunctivitis which, although less common than previously, was still of much moment. Preventive medicine and the advent of the sulphonamides and antibiotics had greatly reduced the incidence and ravages of the disease. In discussing treatment, Dr. Lowth said that frequent irrigation was required for its mechanical effect, and the appropriate bacteriostatic and bactericidal drugs should be exhibited. A culture should be made if possible, and care should be taken to avoid cross-infection. The choice of ointment or drops was a matter of individual taste; ointment had the disadvantage of causing further mechanical impediment to vision. Conjunctival injection was differentiated from ciliary injection by five criteria: (i) the different distribution of the vessels; (ii) the different branching of the vessels; (iii) the movability of the vessels on pressure by a finger on the lower lid; (iv) the brighter colour; (v) extension of the conjunctival vessels.

Dr. Lowth then referred briefly to less important causes of "red eye"—keratitis, scleritis and episcleritis, endophthalmitis and panophthalmitis. He discussed the differential diagnosis and mentioned points in treatment. Finally he set out the broad principles of treatment of the "red eye". He said that in all ophthalmic cases, treatment was generally based on the principles of rest, the local application of heat and local therapy. Rest was usually achieved by the use of mydriatics, and in that connexion he suggested a less frequent use of atropine. Atropine instilled into an eye caused mydriasis, the complete effect of which was not cleared in less than a week, more often 10 days. In a simple case (for example, corneal ulcer) that length of time was not required. In flash conjunctivitis mydriasis was also of value, and usually the patient was fit to return to work in 48 hours. The use of atropine might delay the return for a week longer than was necessary. Dr. Lowth suggested that in those cases one of the weaker mydriatics should be used—for example, homatropine (1% to 2% solution used four times a day). The effect of that drug passed off in less than 24 hours.

Vertigo.

DR. W. A. HOPKINS (Townsville) read a paper entitled "Some Aspects of Vertigo".

Radiation Effects.

DR. ALICE STEWART (Oxford, England) read a paper entitled "Radiation Hazards of Diagnostic X-Rays" (see page 265).

DR. A. G. S. COOPER read a paper entitled "Illustrations and Reports of Late Effects of Radiation" (see page 267).

MR. D. J. STEVENS, Director of the Commonwealth Radium and X-Ray Laboratories, Melbourne, read a paper entitled "The Assessment of Levels of Radiation Doses in Medical Practice".

DR. J. F. GILLOGLEY (Rockhampton) read a paper entitled "Methods of Protection Against X-Rays".

Research in Social Medicine.

DR. ALICE STEWART read a paper entitled "Social Medicine Research Methods" (see page 271).

Obstetrical Hæmorrhage.

PROFESSOR G. SHEDDEN ADAM (Brisbane) read a paper entitled "New Thoughts on Obstetrical Hæmorrhage" (see page 272).

Genital Prolapse.

DR. C. J. MURPHY (Brisbane) read a paper entitled "Genital Prolapse".

Obstetric Experiences in Mackay.

DR. PAUL HOPKINS (Mackay) read a paper entitled "Some Obstetric Experiences in Mackay" (see page 277).

PROFESSOR G. SHEDDEN ADAM congratulated Dr. Hopkins on his paper, and on the painstaking dissection of his private obstetric cases. He said that, taken together with his previously published series of 1500 cases, that detailed analysis of some 2000 private obstetric cases was probably unique in Australia, and was of undoubted value in the common objective—to reduce maternal and fœtal mortality and morbidity to the irreducible minimum, if such there was. Professor Shedden Adam said that his comments would be both of a general and of a particular nature. If, for the sake of discussion, he compared some of Dr. Hopkins' figures with those from the public section of the Brisbane Women's Hospital, it was not because the comparison was a particularly valid one, but because no comparable privately collected series was available. However, it was convenient that Dr. Hopkins' figures were taken of a total of 2000, and that the figures available from the Brisbane Women's Hospital for the year ended June 30, 1957, were taken from a total of approximately 4000 deliveries.

The relatively low incidence of essential hypertension complicating pregnancy shown in Table I (one in 2000) was probably largely a matter of definition. If a blood pressure of at least 140/90 mm. of mercury prior to the twentieth week of pregnancy was taken as a starting point, the Brisbane Women's Hospital figures were 40 per 1000 confinements.

Professor Shedden Adam said that Dr. Hopkins had rightly devoted considerable time to a retrospective study of the cases of eclampsia encountered (six in 2000 or three per 1000). The Brisbane Women's Hospital figures were 2·1 per 1000 deliveries, chiefly unbooked cases. Climatic and seasonal factors in relation to the incidence of eclampsia had always provided an interesting study, and Dr. Hopkins' observations confirmed the impression of a higher incidence during the winter months. For what it was worth, the explanation appeared to lie in the fact that there was less loss of abnormally retained salt through perspiration in the cooler months. In Central and Northern Queensland it might well be that salt loss through perspiration in the hot summer months had conditioned the population to habits of high salt intake in their food, and that habit might not be automatically given up during the winter, with harmful consequences to some pregnant women. Dr. Hopkins had emphasized two important points in regard to eclampsia. The first was the difficulty in foretelling which patients with severe preeclamptic toxæmia were at risk with regard to developing the convulsive phase. Apart from the evidence of cerebral dysrhythmia as shown in the electroencephalogram (hardly a point of general clinical application), perhaps the patient's physical habitus (the sthenic type) and perhaps the family history, there was probably no way of prognosticating with accuracy. It was probably for that reason that, apart from the prophylactic aspect of the eclamptic problem, particular emphasis was now being given to the condition of imminent eclampsia. In other words, all patients with severe preeclamptic toxæmia were potentially at risk in regard to actual eclampsia, and each obstetrical centre had its own minor preferences with regard to immediate therapy. Some pinned their faith on controlled intravenous or intramuscular injections of hypotensive agents in full dosage. Carey of Auckland reported some success with protoveratrine A ("Puroverine"), 0·2 to 0·3 mg. every three or four hours, and for actual eclampsia he used a 5% glucose solution containing 1·0 mg. to the pint, given intravenously at a rate of 40 drops per minute, until the blood pressure fell, then slowed down. Others, including Brisbane workers, centred their treatment of imminent eclampsia around a combined sedative and hypotensive agent, tribromethanol ("Avertin"), given by the rectal route as a 3% solution in distilled water at 40° C. The average dose for a ten stone patient was 5·5 ml. of "Avertin" in 223 ml. of water. It should be tested with Congo Red before use, to exclude toxic degradation products from overheating. The dose might be repeated in four to six hours. The object of any such treatment of severe preeclamptic toxæmia was to bring the patient into a more suitable condition to withstand the inevitable stimuli arising from the next stage, which was the emptying of the uterus. That emphasized Dr. Hopkins' second point, namely, the extent to which a severely preeclamptic patient's condition might deteriorate during labour. Delivery by Cæsarean section might be preferable for a primigravida with a closed cervix and a high fœtal head, or for a parous woman with a history of previous dystocia or protracted labour. Fortunately many eclamptic patients went into labour soon after entering the convulsive phase, and in parous women at least simple artificial rupture of the membranes was often sufficient to accelerate delivery.

Professor Shedden Adam then said that he would like to comment briefly on one item in Table III of Dr. Hopkins' paper, a problem that worried them all—the unexplained still births, of which there were eight in 32 still births. Routine autopsy of all but macerated fœtuses would, of course, reduce that figure somewhat; but with all facilities, the real reason for some of those unexplained still births still eluded them. Undoubtedly the cause was physiological rather than pathological, and was concerned with the all-important subject of fœtal oxygenation and placental insufficiency, concerning which much investigation was proceeding. In the meantime, the early recognition and prompt treatment of hypertensive and toxæmic states during pregnancy, and a careful assessment of each case of apparently prolonged gestation, would keep that figure down to a minimum. As a matter of interest, he noted that the still-birth rate in Dr. Hopkins' series of 2000 cases was 16 per 1000, which would compare with a figure of 14 per 1000 for the public section of the Brisbane Women's Hospital.

The corresponding figures for the neonatal death rate was 11·5 per 1000 (Dr. Hopkins' series) and 13 per 1000 for the Brisbane Women's Hospital, including non-viable fœtuses whose mothers had miscarried at the Brisbane General Hospital. The consistency with which prematurity figured as a major cause of neonatal death in all such series was confirmed by Dr. Hopkins' finding that 10 out of 23 such deaths were due to prematurity. The Brisbane Women's Hospital figures were very similar, and Mary Crosse stated that prematurity was seldom responsible for less than 50% of neonatal deaths. Now that adequate care of the premature neonate was becoming increasingly available throughout the country, the onus was on the obstetrician and the medical sociologist to reduce if possible the incidence of premature labour in the community—a figure which remained very constant at approximately 5% of all births. Professor Shedden Adam said that that subject was being studied in his department.

Professor Shedden Adam finally said that he would like to make some useful comment on the subject of congenital malformations as a cause of fœtal loss; but in actual fact, in spite of their experience with rubella, they knew very little about those errors of development, and much further research was needed. In conclusion, he said that the value of such statistical surveys depended on the background of experience and wisdom with which they were interpreted. In listening to Dr. Hopkins' paper, it was obvious that he had those qualities of experience, humility and wisdom which made such a survey worth while.

Heart Turns.

SIR ALEXANDER MURPHY (Brisbane) read a paper entitled "Heart Turns".

Hæmoptysis.

DR. E. SILBERSTERN (Westwood Sanatorium, Queensland) discussed hæmoptysis. He said that he had discovered 52 diseases of which hæmoptysis might be an integral part. In making a diagnosis, therefore, the first requisite was to confirm that the substance in question was blood, and the second to make certain that it originated from the respiratory tract, from the larynx downwards. Generally, fresh blood from the lung was bright red and frothy, venous blood darker red and frothy, old blood even darker. Blood originating from the stomach was as a rule altered by hydrochloric acid and might contain food residues. It should be remembered that blood from the upper part of the respiratory tract, from the nose or naso-pharynx, from teeth, etc., might trickle down the throat and be coughed up, that an aneurysm of the aorta might burst and cause bleeding into the trachea or œsophagus, and that œsophageal varices might burst or bleed, the blood being swallowed or inhaled and returned by vomiting or coughing or both. The extent of the hæmoptysis was as a rule determined as (i) minimal, (ii) light, (iii) medium or (iv) severe. Minimal hæmoptysis consisted of isolated specks or streaks of blood; it might be the harbinger of a larger hæmoptysis. Light hæmoptysis meant the loss of up to half a fluid drachm of blood (not of sputum); in that connexion it was important to bear in mind the great staining property of hæmoglobin. A medium hæmoptysis meant the loss of half a fluid drachm to three fluid ounces, and a severe hæmoptysis the loss of three or more fluid ounces.

Dr. Silberstern then went on to discuss hæmorrhage from the lower part of the respiratory tract (from the larynx downwards). He said that causes might be (i) trauma, (ii) pathological conditions of the circulatory system, (iii) diseases of the blood and blood-forming organs, (iv) infections of the respiratory tract itself, (v) newgrowth. In the experience of the Chest Clinic of Rockhampton, the most common causes of hæmoptysis were, in order of importance, (i) bronchiectasis, (ii) pulmonary tuberculosis (both initial and advanced stages), (iii) tumours, particularly carcinoma, (iv) pneumonia and other infections of the lungs, (v) diseases of the circulatory system. However, it should never be forgotten that, even if an explanation for the hæmoptysis was found, another hæmorrhage-causing condition might coexist. Conditions which might obscure the diagnosis and simulate an hæmoptysis were (i) diseases of the nasal organs (epistaxis, naso-pharyngeal hæmorrhage, adenoids, diseases of the sinuses), (ii) mouth disorders (treatment or extraction of teeth, conditions of the tooth sockets, sucking of blood from the gingiva, conditions affecting the salivary glands), (iii) conditions affecting the tonsils or tonsillar beds after tonsillectomy, traumatic conditions of the larynx or laryngo-pharynx, foreign bodies in the œsophagus, and œsophageal varices, particularly in association with diseases of the liver and hæmatemesis.

Discussing prognosis, Dr. Silberstern said that it had to be guarded, unless full investigation revealed an innocent cause for the hæmoptysis. Treatment varied according to the degree of hæmoptysis. In minimal hæmoptysis, reassurance was required; but the patient's activities should be restricted and he should be closely observed. In light hæmoptysis, the patient should be reassured, but put on full bed rest, as it might be the forerunner of a severe hæmorrhage. If the situation was such that treatment had to be given, intramuscular injections of calcium gluconate

(5 to 10 ml. of a 5% to 10% solution), or of vitamin K (10 mg.) were not unreasonable, even if laboratory investigation revealed no lack of calcium or vitamin K. If the hæmoptysis was of medium severity, the patient should still be reassured, but it should be regarded as a serious sign. Full bed rest with absolute prohibition of even the slightest activity was imperative. One-sixth of a grain of morphine should be given by injection at once, both as a sedative and in order to restrict an exaggerated cough reflex to the physiological necessity. Soiled bed linen should be covered with towels, and a bowl put conveniently for further expectoration. An ice cap might be applied if desired. A light diet and cool or lukewarm drinks might be given. Ice cubes had no value, but could be given. The patient should have no excitement and no visitors. The foot of the bed should be raised six to 10 inches. Calcium gluconate or vitamin K might be injected. Blood transfusion, vasomotor regulation with "Novocain", artificial subcutaneous emphysema and artificial pneumothorax were not recommended. In a few cases thoracic surgery might be considered. Dr. Silberstein said that he had had no personal experience with "Reptilase", "Sangostop" or toluidine blue; good results with the two last-mentioned had been recorded elsewhere. Dr. Silberstein finally discussed the treatment of severe hæmoptysis, which he said was very serious, and might be fatal in a few minutes. The patient should be kept at absolute rest in bed in the position of greatest comfort. An injection of one-sixth of a grain of morphine should be given immediately, and the patient should be reassured and his bed surrounded by screens. Vitamin K should be given by intramuscular injection; it was preferable to calcium gluconate. Sedatives should be given as required, and the foot of the bed raised six to 10 inches. Oxygen might be required if the patient struggled for air. The patient should be subjected to no effort at all, not even the making of his bed. Treatment, if it was possible, was as had been described. Death usually resulted less from loss of blood than from the patient's drowning in blood, which filled the bronchi, bronchioli and alveoli and blocked the air pasages and breathing surfaces.

Lung Cancer.

DR. T. G. PAXON (Cairns) read a paper entitled "The Future Incidence of Lung Cancer".

Emphysema.

DR. R. A. DOUGLAS read a paper entitled "Emphysema" (see page 279).

Preventive Medicine in Pædiatrics.

DR. I. BRODY (Gordonvale) read a paper entitled "Preventive Medicine in Pædiatrics" (see page 281).

Allergic Manifestations in the Respiratory Tract.

DR. P. A. EARNSHAW (Brisbane) read a paper entitled "Allergic Manifestations in the Respiratory Tract".

Keratoacanthoma.

DR. W. R. HORSFALL (Cairns) read a paper entitled "Keratoacanthoma".

Fluothane.

DR. W. H. J. COLE (Melbourne) read a paper entitled "Fluothane".

Small Bowel Dyspepsia.

DR. K. J. J. DORNEY read a paper entitled "Small Bowel Dyspepsia".

Diseases of the Biliary Tract and Pancreas.

A symposium on diseases of the biliary tract and pancreas was presented by DR. L. I. BURT (Brisbane), DR. I. A. LESTER (Cairns) and DR. B. CAVATE (Rockhampton). Dr. Burt's paper was entitled "Internal Drainage of the Common Bile Duct" (see page 282). Dr. Lester's paper was entitled "Gall Bladder Disease: Symptoms and Differential Diagnosis" (see page 284).

Carpal Tunnel Syndrome.

DR. G. A. DOUGLAS (Townsville) read a paper entitled "Carpal Tunnel Syndrome".

The Crippled Child.

DR. U. LEY (Townsville) read a paper entitled "The Crippled Child".

Trade Exhibition.

During the Conference a trade exhibition was held. The Exhibitors included the following: Andrews Laboratories Pty. Ltd., Boots Pure Drug Co. (Australia) Pty. Ltd., Burroughs Wellcome & Co. (Australia) Ltd., Drug Houses of Australia, D.H.A. (N.S.W.) Pty. Ltd., Parke, Davis & Co. Ltd., Schering Pty. Ltd., The British Drug Houses (Australia Pty.) Ltd., Warner-Lambert Pty. Ltd., Johnson & Johnson Pty. Ltd., May & Baker (Australia) Pty. Ltd., Merck Sharp & Dohme (Australia) Pty. Ltd., W. Ramsay (Surgical) Ltd.

British Medical Association.

VICTORIAN BRANCH: SECTION OF PREVENTIVE MEDICINE.

A MEETING of the Section of Preventive Medicine of the Victorian Branch of the British Medical Association will be held on Thursday, September 10, 1959, at 4.30 p.m., at the Medical Society Hall, 426 Albert Street, East Melbourne. Dr. Alan Ferris, of the Epidemiological Research Unit, Fairfield Hospital, will speak on "Enteric Viruses in a Semi-Closed Community". All interested are invited to attend.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE NEW ZEALAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION AND THE AUSTRALASIAN MEDICAL GAZETTE.[1]

[From the *Australasian Medical Gazette*, November, 1896.]

A BRANCH of the British Medical Association has been inaugurated in New Zealand. With the advent of the year 1897 the New Zealand Medical Journal will cease to appear, and becomes incorporated with the Australasian Medical Gazette, still retaining the valued services of its Editor, and representing the New Zealand Medical Association. We congratulate our New Zealand confrères upon their determination to adhere to the principle involved in the consensus of opinion expressed at the last Session of the Intercolonial Medical Congress of Australasia held at Dunedin, "that all the colonies should unite in having one strong journal to represent the interests of the profession in the Australasian colonies".

Correspondence.

A FRYPAN AS A STERILIZER.

SIR: I have found that the "Sunbeam" electric frypan with a glass lid makes an excellent hot-air sterilizer. The low initial cost of the frypan, plus its modest size compared with conventional sterilizers, makes it a very convenient unit for the general practitioner's surgery.

A metal grid with feet approximately a quarter of an inch long should be placed in the frypan to prevent the material being sterilized from touching the bottom of the pan; the grid also allows free circulation of air. A small thermometer such as the "EMil GS10687" is placed on the grid in such a manner that the scale may be read through the glass frypan cover. Satisfactory sterilization is obtained by maintaining a temperature of 160° C. for one hour.

"Sunbeam" frypans are calibrated in degrees Fahrenheit, and a new unit will reach a base temperature close to 216° C. when the dial is set for maximum heat. A frypan that has been in use for some considerable time may be unable to reach the required temperature because of

[1] From the original in the Mitchell Library, Sydney.

wear on the thermostat contact points. In this case mechanical readjustment will be required.

The Sunbeam Corporation have advised that any Sunbeam Service Depot will willingly make any necessary adjustments to ensure that the frypan gives efficiency in this field.

Yours, etc.,

R. E. DAVIS, F.I.M.L.T., F.I.S.T.
Department of Hæmatology,
Royal Perth Hospital,
Perth, W.A.
July 31, 1959.

SKIN STERILIZATION.

SIR: A shock assault by enterprising commercial gentlemen is being made at present upon our hospitals in an effort to impose the use of their products for skin sterilization. It is therefore of interest to note recent research on hand-sterilization in the Surgical Clinic of the Free University of Berlin, in cooperation with the famous Robert Koch Institute, reported at the annual meeting of the German Surgical Society, held last year in Munich.[1]

These workers tested hexachlorophene, a quarternary ammonium compound and alcohol. They found that under the conditions of the operating theatre, as well as in the laboratory, alcohol was by far the best. They write: "We can declare that to-day, as in the past, alcohol is still the unsurpassed method of hand-preparation." After the hands are washed with soap and water and brush for two minutes, and dried with a sterile towel, they are placed in alcohol (80%) for five minutes.

The publications reporting astonishing results with hexachlorophene are described as misleading, and unable to withstand critical examination. They give other references where this preparation can be factually evaluated. They advise that the much vaunted chemical protective mantle, which hexachlorophene is supposed to confer upon the skin, should not be overrated, for in spite of it, "a high percentage of pathogenic cocci are found, not to mention the fact that gram-negative flora are practically uninfluenced".

In March, 1959, in the same journal, Boheimer of London suggested that these authors had overlooked the increasing effectiveness claimed for hexachlorophene after repeated use. But the authors replied that they had not overlooked this factor, but had carried out their comparative investigations after three years' regular use of hexachlorophene.

It is sometimes said of alcohol that its effect is only transitory, ending with its evaporation, so that skin organisms from the depths soon reinfect the surface. On the other hand, Göpel and his colleagues write: "According to our experience, disinfection is so established with alcohol, even in the depths of the skin, that after operations averaging 2½ hours, no rise worthy of mention was observed in the bacterial count. This result is possibly due to a hardening-fixation of the skin, through which is also explained the lessening of the fluid in the gloves, noted in our experiments."

Yours, etc.,

VICTOR J. KINSELLA.
235 Macquarie Street,
Sydney.
July 25, 1959.

MEDICINE AND ATOMIC ENERGY.

SIR: Medical practitioners realize more vividly than the average citizen that a world war fought to a finish with hydrogen bombs would mean the end of civilization as we know it.

We read much about the conflict of ideologies, the balance of power, the influence of nationalism and all the other factors in this complicated global drama, but we know that there is now no adequate protection for humanity except world government. The principle is simply expressed by one writer as follows:

> We are all familiar with municipal government for municipal affairs and state government for state affairs. A further progressive step is made when the various states of one nation agree to federal government for federal affairs. The final extension of the system of law and order will be world government for world affairs.

The methods by which various obstacles may be overcome are described in many well-known books. Though the British Medical Association is a non-political organization, we, as guardians of the physical and mental health of the people, would do well to express our official approval of the principle of world government. Can any greater example of preventive medicine be imagined than the prevention of a world war in this atomic age?

Yours, etc.,

VICTOR H. WALLACE.
61 Collins Street,
Melbourne.
August 1, 1959.

TETANUS PROPHYLAXIS.

SIR: Recent letters in your Journal have thown considerable doubt on the efficacy of anti-tetanus serum, but few of us will have the courage to refrain from using it, because of the fear of permitting our patients the risk of a deadly malady, with its evil prognosis and probable ensuing litigation. And now Dr. Hunter, in a most persuasive letter in the issue of July 18, 1959, indicates that almost identical risks are incurred if the serum is used.

May I suggest, Sir, than an authoritative statement be made by a panel of experts to assist those perplexed persons who find themselves perched precariously between the frying pan and the fire?

Yours, etc.,

M. A. O'HALLORAN.
19 Gerrale Street,
Cronulla, N.S.W.
August 3, 1959.

ANKYLOSTOMIASIS OR ANCYLOSTOMIASIS?

SIR: Would you allow me to seek the views of readers in regard to the term ank(c)ylostomiasis?

This word in the notifiable diseases of the Journal is spelt with a "c". However, the "British Encyclopædia of Medical Science" chooses "k".

In my experience the word has always appeared in writing with "k" and has always been pronounced with a "hard c".

If an analogy is accepted from the English word "cycle", which is derived from the Greek word "*κύκλος*", then the word in question will have to be pronounced "an-s-ylostomiasis", unless one refuses to accept any logical nexus between the spelling and the pronunciation of words. There certainly is a similarity between "*κύκλος*" and "*αγκύλος*" which gives justice to this latter pronunciation.

My aim here is to discover whether members of the medical profession accept this pronunciation. Otherwise it seems to me that if the word is pronounced with a "hard c", then it must be written with a "k".

Yours, etc.,

LESLIE J. SHORTLAND.
"Otaki",
Marrickville.
July 30, 1959.

AUTONOMIC DYSPRAXIA.

SIR: In a letter published in the Journal of July 4, Dr. Bryan Haynes goes to some length to explain the sudden collapse and death of a man at police headquarters and the death of the space monkey, Able, as classical examples of what he defines as autonomic dyspraxia.

The relation of widespread dysfunction to emotional stress has been known since the early days of medical history, and it is generally known that the autonomic nervous system is concerned in the mediation of impulses resulting in changes of vascular tone and function of glandular and other internal organs which are not under voluntary control. The effects of stress on man have been

[1] Göpel, H., Rücker, G., and Schütz, W. (1858), *"Zur präoperation Händedesinfektion", Chirurg.*, 10: 433 (October).

the subject of considerable interest in recent years, and the remarkable work of Selye and others has shown the complexity of this problem.

The hypothalamus is undoubtedly concerned with regulation of the autonomic system, but the precise afferent and efferent connexions of it are still imperfectly understood. It is dangerous to violate facts by trying to make them fit into simple schemes. The concept of man's physiology and pathology revolving around his hypothalamus is indeed incomprehensible to our generation, because it is an unscientific and uncritical over-simplification of complex physiological mechanisms and can be described as a regression to the days when the pineal was considered to be the seat of the soul.

Yours, etc.,

GEORGE SELBY.

Hengrove Hall,
193 Macquarie Street,
Sydney.
July 30, 1959.

NEURO-PSYCHIATRIC COMPLICATIONS OF MECAMYLAMINE THERAPY.

SIR: I have read with interest the report of "Neuro-Psychiatric Complications of Mecamylamine Therapy" by Dr. Murphy and Dr. Sutherland. The case they report has many features in common with the cases reported by Neilson and I,[1] and by Schneckloth *et alii*.[2] Neilson and I were uncertain whether this syndrome could be properly attributed to mecamylamine, since withdrawal of the drug in one patient did not alter the neurological state, whilst in the other patient autopsy revealed punctate hæmorrhage in the region of the basal ganglia. The symptoms suffered by the patient reported by Dr. Murphy and Dr. Sutherland seemed to have disappeared when the drug was withheld. It seems reasonable therefore to attribute them to the mecamylamine.

Since reporting these cases three years ago, I have not encountered any similar manifestations. I have refrained from using the drug where there has been a past history of psychosis or clear evidence of cerebral vascular disease. It seems that, providing these precautions are observed and that large doses of the drug are avoided, this syndrome is uncommon.

Yours, etc.,

A. E. DOYLE.

Department of Medicine,
University of Melbourne,
The Royal Melbourne Hospital,
Parkville, N.2, Victoria.
August 3, 1959.

THE DRIVER IN RELATION TO THE PHYSIOLOGICAL LIMITATIONS IMPOSED ON HIM AS A HUMAN BEING.

SIR: I would refer to the above Special Article (MED. J. AUST., July 4, 1959). The writer of this article develops and elaborates the theme of how the brain comes to "neglect" repeated stimuli of even intensity, conveyed from the various sensory end organs. In fact, this is the essential basis of a three-page paper.

I beg to submit that such cortical neglect tends to be relative and selective rather than absolute, and should not normally be a cause of traffic misdemeanours or accidents. I protest against the practice of taking a well-accepted physiological principle and applying it in such a way that it may no longer be true, logical, or of practical importance in its new context. For example, the article contends that a driver, having looked at many and various red lights (including car tail lights, road repair lights and advertising lights), can for that reason be pardoned for not "seeing" a red traffic-light signal at an intersection. Surely this is carrying physiology to the point of absurdity! Would it not be equally logical to say that a taxidriver, having seen many pedestrians during the day, may be excused for running down two or three of them, because, due to the repeated "pedestrian stimulus" his brain has come to neglect them?

Again, in the discussion of visual fields, a driver is exonerated from liability for not seeing a speed limit sign on the left of the road, because he was concentrating on the road itself. What really happens is this. In man the central part of the visual field is adapted for keen vision and the peripheral field for grosser uses. The peripheral field is also sensitive to sudden, unexpected movements. A good driver does not concentrate his vision exclusively on the middle of the road. His eyes move and he scans the field of vision. If, for example, a road sign is picked up by his peripheral vision, he then has a quick look at it with his central vision to read the details. Any wartime fighter pilot will confirm that this constant moving of the eyes is very necessary. If a driver feels that he cannot take his eyes off the road long enough to read a speed limit sign, then it would seem that he is either driving too fast for safety, or that there is some pathological condition present.

The Special Article says that "accommodation has to deal with the ability of the eye to vary the amount of the pupil exposed. This is brought about by the contraction and relaxation of the iris". In actual fact, accommodation is the mechanism whereby the converging power of the eye is increased so that a distant image is still retained, when the focus is changed from far to near. It is then that the pupil does contract on accommodation, but this is not due to the presence or absence of light in this particular case. The writer of the article appears to be quite confused in his ideas of accommodation, light reflex, dark and light adaptation and even, perhaps, presbyopia. He speaks as if these were all part of accommodation.

My chief criticism of the article, however, is directed against what I consider to be incorrect application of physiological principles. It is like selecting a proposition that is true and valid *in vitro* under laboratory conditions, and asserting it to be equally true and valid *in vitro* for all conditions.

Yours, etc.,

FERGUS N. YEATES.

Ballow Chambers,
Wickham Terrace,
Brisbane, Queensland.
July 31, 1959.

[1] *Brit. Med. J.*, 1956, 2: 1431.
[2] *J. Amer. med. Ass.*, 1956, 162: 868.

Obituary.

CHARLES NORMAN PAUL.

WE are indebted to Dr. O. A. Diethelm for the following account of the career of the late Sir Charles Norman Paul.

> Knowledge and wisdom far from being one,
> Have oft times no connection. Knowledge dwells
> In heads replete with thoughts of other men;
> Wisdom in minds attentive to their own.
> Knowledge is proud that he has learned so much,
> Wisdom is humble that he knows no more.
>
> —COWPER.

When I read these lines of Cowper, applicable to many great men well versed in their lore but filled with humility, I thought how well they were exemplified in our late friend and colleague, Sir Norman Paul, M.B., Ch.M., F.R.A.C.P., who was taken from our midst suddenly, while enjoying comparatively good health and fully occupied with his life's work, which he loved so much. He died in harness as he desired.

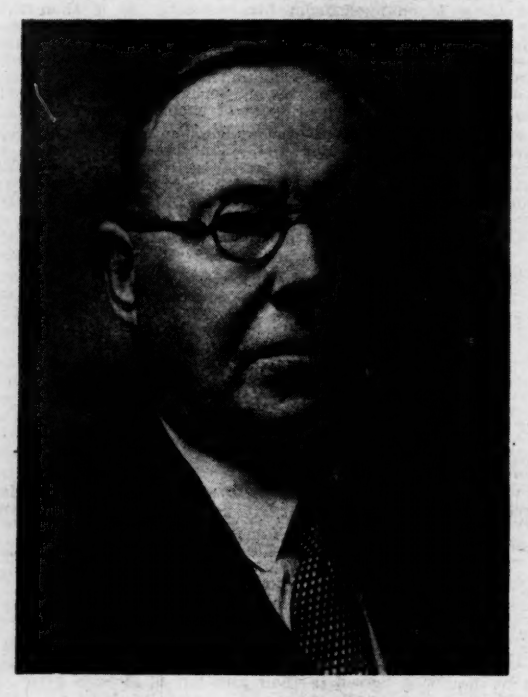
Charles Norman Paul was born in Camperdown (Sydney), on March 6, 1883, his parents being Mr. and Mrs. John Paul, later of Strathfield, and still later of "Danmark", Wolseley Road, Point Piper. There he himself lived until he built his beautiful home at "Lorraine", Dalley Avenue, Vaucluse, just after his marriage in 1914. He was the fifth of Mr. and Mrs. John Paul's six sons, all of whom were professional men, two being medical men, two doctors of dental surgery and one a doctor of science. One son, Dr. Albert Paul, died in 1916, on service in France during the Great War. Only one is now alive, Dr. Oscar Paul, for some years retired; he was one of the first specialists in orthodontal dental surgery.

Norman Paul received his secondary education at the Sydney Grammar School. He did not proceed to the university immediately after the termination of his secondary education at the Sydney Grammar School. It was his primary intention to take up a commercial career, and he joined the business of his father, who founded the firm of Paul and Gray, ship chandlers, in Sydney. He showed great promise in this career; but after a few years the professional urge was too strong, and he decided to do a medical course. He entered the University of Sydney,

where he graduated in medicine in 1910 with the degrees of bachelor of medicine and master of surgery.

After graduation he was a resident medical officer at the Royal Prince Alfred Hospital, Camperdown, from December, 1910, to March, 1911, acting as junior pathologist and relieving resident physician and surgeon. There he worked under his great friend Sir Herbert Schlink, who was the chief resident medical officer at the institution during that period. This gave him his first insight into the practical application of pathology, to which he devoted many years of close post-graduate study in London and Vienna, and later in Sydney, as a specialized adjunct to his dermatological studies and work.

In 1911, while a resident medical officer at Royal Prince Alfred Hospital, he decided to go abroad and do post-graduate work, mainly in the speciality of dermatology. He first pursued his studies on syphilis and skin diseases in Vienna, which at that time was the centre of the post-graduate field and the Mecca of all graduates in medicine. He attended the *Krankenhaus Allgemeines* for six months,



and received his diploma from the University of Vienna for his work there.

He proceeded to London in 1912 for further studies in dermatology, and spent a year in that great city of medical culture and learning. During that period he was first clinical assistant at St. Bartholomew's Hospital to Dr. Adamson, and worked at St. John's Hospital for Diseases of the Skin in Leicester Square, and later attended King's Cross Hospital and the Great Northern Central Hospital, working with Dr. Whitfield, and also the London Hospital in the clinic of Dr. Sequeira. With these three world-renowned specialists and dermatologists he developed a very close friendship.

In 1913 he returned to Sydney and commenced consultant practice as a dermatologist, which he continued for 46 years till the day of his death. He was one of the first, if not the very first, to use radium and later X-ray therapy in his practice. In this connexion one cannot fail to mention Sister Sparkes, who gave him invaluable assistance particularly in the use of radium, and his secretary, Mrs. Wellman, both of whom, at the time of his death, had been in his service for 32 years.

In 1914, he married Louise Morell, the daughter of J. B. Morell of Orange. Norman Paul's home was his castle; he was ever watchful to see that nothing was left undone that would contribute to his wife's happiness and comfort, for which no sacrifice was too great. On her side, Lady Paul was a wonderful helpmate to her husband, in both his professional and his social activities. She was his constant inspiration during the whole of their married life. Together they built up their lovely home in Vaucluse, and gradually added valuable pictures and paintings, antiques and furniture. It is a real home in every sense of the word, and Norman Paul was justly proud of it.

In 1914 he was appointed to the honorary dermatological staff of Sydney Hospital, and he worked in that capacity until he retired in 1943, having reached the age limit of 60 years. He remained a consultant honorary dermatologist till his death. In 1916, he was appointed honorary dermatologist at the Royal Alexandra Hospital for Children at Camperdown, and held that position for sixteen years, until he retired from that service. In the same year he was appointed honorary dermatologist to the Renwick Hospital for Infants and also to the Royal North Shore Hospital. In 1932, on his retirement from the Royal Alexandra Hospital for Children, he was made honorary consultant dermatologist.

In 1937 he was awarded the Coronation Medal, and in the same year he was created a Knight Bachelor, in recognition of his work in the field of dermatological science. He was president of the New South Wales Branch of the British Association of Dermatology, and in 1938 he was made an honorary member of that association—at that time the only recipient in the British Empire outside the British Isles. He was also made a life governor of Sydney Hospital and of the Benevolent Society of New South Wales. In 1938 he was appointed simultaneously director and president of the Board of Sydney Hospital, positions which he retained till his death. In that year also he was made a Foundation Fellow of The Royal Australasian College of Physicians. In 1941, on the appointment by the Government of a Hospitals Commission for the control and management of hospitals in New South Wales, Norman Paul was made a member and vice-chairman, positions which he energetically and enthusiastically filled in a most unselfish way for a period of three years until he retired in 1944.

Norman Paul's influence on dermatology was marked by various publications in dermatological and other medical journals, especially by the publication of two books on skin diseases, which received world-wide acclaim and recognition—"The Influence of Sunlight in Production of Skin Cancer" and "Cutaneous Neoplasms". He was one of the first dermatologists, probably the first, to recognize the dire effect of the sun's rays as an ætiological factor in skin cancer. For this work alone he was congratulated by dermatological associations and authorities all over the world. He made several visits overseas to the United Kingdom and Europe, on which he always took the opportunity of visiting and renewing friendships with such doyens in the dermatological world as Adamson and Whitfield.

Norman Paul was very fond of motoring, and particularly when on holidays or travelling, took a keen delight in driving himself. He was also a first-class billiards player; he took this pastime seriously as a young man and was coached by a well-known professional, Hooper. He could have won a New South Wales amateur championship in his younger days if he had set his mind on it and could have afforded the time to practise. In later years, he was handicapped by his eyesight, and by lack of time to practise. At the time of his death he was a member of the Australian Club, the Royal Sydney Golf Club and the Royal Automobile Club, and for a number of years he was a member of the Australian Jockey Club.

Although it is difficult to allocate one sphere alone in which Norman Paul and his work will be most remembered, perhaps it is his monumental work and unselfish devotion in the advancement of that great public institution, Sydney Hospital, both as a public hospital and as a clinical teaching hospital attached to the University of Sydney. During his period as president of the Board, great advance was made, not only in building and adding new wards and remodelling old wards, but also in providing accommodation for the vastly increased nursing staff, necessary to cope with the additional work, in providing increased bed capacity and increased resident and honorary medical staff, and in the equipment of new special departments to meet all progressive hospital needs. He was not in favour of Sydney Hospital's being moved; he was of the opinion that it would remain in its present position for many years, but that if a change was made, at least a casualty and accident clearing station should still remain, to meet emergency conditions. With this end in view, large sums of money were spent in renovations and additions to the present hospital, with the provision of all necessary modern equipment for the medical and nursing staff.

Seneca once said "There is no day without sorrow". Although Norman Paul's life was one of happiness in his home and satisfaction in his work, he had one great day of sorrow, September 23, 1921, when his younger brother, aged 36 years, a promising ophthalmologist in Sydney on the staff of Sydney Hospital, was killed in a motor accident. This was a terrible blow to Norman, and left its impact on him for a very long time; the brothers were extremely close to each other. After this tragic occurrence Norman took the place of his brother, who had been my close friend since University days, and he and Lady Paul became intimate friends of my wife and myself.

Norman Paul's genial presence will be sadly missed by his colleagues and friends, not only in the dermatological field of medicine, but also because of his devotion to the work of Sydney Hospital. He was a great humanitarian and a man of the highest principles, yet always humble. His aim was to add what he could to life, not to get what he could from it. Important as these characteristics are in the life of a medical man engaged in a busy practice, I would rather speak of his innate qualities as truly one of nature's gentlemen. He had a lovable and most unselfish nature, never counting the cost, but doing good deeds unostentatiously for the pleasure they afforded him. He was a man of few words except when the occasion demanded, and then he was at his best. He never said anything, or esteemed anything of advantage, that would make him break his word in the slightest degree or lose his self-respect. Characteristic of him were his dignity and his humility. Although he had a charming personality, he was really of a very shy nature; but the better one got to know him, the more admiration he commanded. He led a strenuous life, both in his home and in his profession, apart from his Sydney Hospital activities, but it was all well organized. He had a great faith, than which there is nothing more wonderful in life—faith in the future of his profession and of Sydney Hospital and in himsef—and he resolutely and courageously carried out what he had satisfied himself was just and necessary and in the best interests of the public.

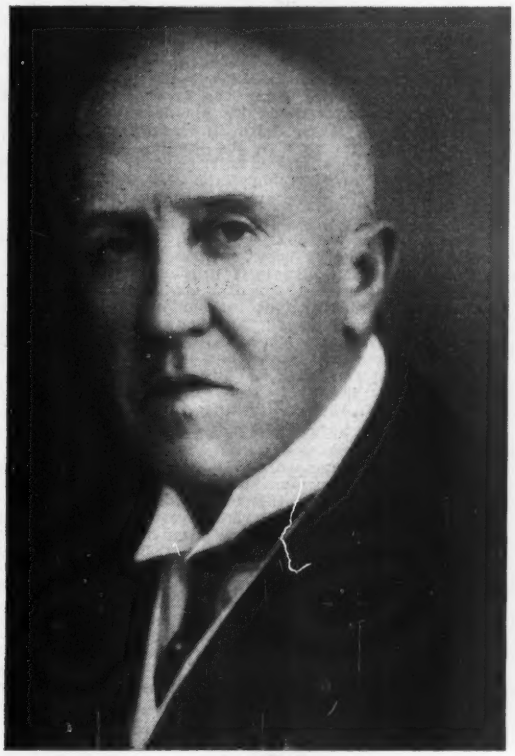
Apart from his happy home life, if there is one quality which could be abstracted from a full list that would characterize the life and career of Norman Paul, I would say it was his intense loyalty to his friends. In the life of a young man the most essential thing for happiness is the gift of friendship, and all through his long life and career this was well illustrated. He was as dependable as the Rock of Gibraltar, and with this attribute he combined a simplicity of nature which all who knew him admired. He loved children and also had a love for animals and birds of all kinds. I can speak with gratitude of his kindness to our four sons, all of whom he had known since birth, and who all loved him.

On Friday, May 22, he suffered a cerebral lesion while at work in his consulting rooms, and he died at his home three days later. We must now ring down the curtain of his life. He was a great man and a great example, and the memory of a great name and of a great example is the legacy of heroes. Nothing that was worthy in a man's life departs, no truth or goodness realized by man ever dies or can die. Norman Paul's memory will live long, and his good deeds and the devoted life he has led must surely earn him his eternal reward. G. K. Chesterton has said: "If seeds in the black earth can turn into beautiful roses, what might not the heart of man become in its long journey towards the stars?" Norman Paul has now made this journey, and I, with all his friends, have no fear that he has found peace. To his wife, to his only surviving brother and to the various members of their families, we offer our sincerest sympathy in their great loss.

GEORGE MONCRIEFF BARRON.

THE death of Dr. George Moncrieff Barron in May, 1959, brought to a close a full and happy life of service; particularly his work for children will be long remembered. George Barron was born in Edinburgh on December 2, 1876, the son of the Reverend Andrew Barron and Mrs. Barron. His father, although an ordained minister, was a schoolteacher. The family migrated to Australia in 1886, and the Reverend Andrew Barron became the minister of the Grafton Presbyterian Church. In Grafton George Barron attended a school conducted by his father; among his school mates were two boys whose names later became well known—Earle Page and Iven Mackay. On leaving school at the age of 16 years, George Barron entered the New South Wales State Public Service, and worked in the Treasury for 10 years. Having saved enough money, he then began the study of medicine at the University of Sydney, graduating with the degrees of bachelor of medicine in 1908 and master of surgery in 1909. After having served for a year as a resident medical officer at Sydney Hospital and for four years as surgeon at Macleay District Hospital, he began private practice in Manly, where he remained for the rest of his life.

George Barron was a loyal member of the British Medical Association; he was a member of the New South Wales Branch Council from 1932 to 1946, and became president of the Branch in 1939. He was admitted as a Fellow of the Royal Australasian College of Surgeons in 1933. From 1941 to 1946 he was a member of the New South Wales Medical Board.



As a trusted family doctor, George Barron will long be remembered in Manly. However, undoubtedly his most outstanding work was done in connexion with the Far West Children's Health Scheme. The story of this wonderful piece of social service initiated by the Reverend Stanley Drummond and his wife in 1924 is too well known to need to be told again here. George Barron threw his weight behind it enthusiastically from the very first, and in 1926 became the medical superintendent of the scheme. With Stanley Drummond he set up the home in Manly in 1929, which since then has offered specialized services to 8000 sick and handicapped children. The establishment and activity of the country health services all over New South Wales undertaken by the Far West Council owed much to his guidance as chairman of that council. These services have helped more than 60,000 children. He remained chairman until 1958, but even after his resignation he continued to work for the scheme. Although he vacated the position of medical superintendent when he was over 70 years old, he continued to visit the Far West Home practically every day almost until his death. In 1937 His Majesty the King created him an Officer of the Most Excellent Order of the British Empire, in recognition of his work for the children of the Far West.

George Barron was interested in sport, and was an active member of the Manly Golf Club, in which he held several offices, the Manly Lawn Tennis Club, of which he was president from 1920 to 1954, and of the Manly Surf Club. He was also a follower of cricket.

It is not surprising that he should have been associated with Rotary; he was a member of the Manly Rotary Club for many years. He was keenly interested in his church, and for many years was an elder of St. Andrew's Presbyterian Church, Manly. There his funeral service was held on May 19, and the packed church was an eloquent tribute to the affection and esteem in which he was held. The service was a triumphant close to a life of dedication and the triumph was fittingly expressed by the playing of Handel's "Hallelujah Chorus" on the organ. The memory of this will undoubtedly bring comfort to his widow and family in their personal loss.

THOMAS GILLIS WYNNE.

WE are indebted to Dr. Raymond Hennessy for the following account of the career of the late Dr. Thomas Gillis Wynne.

Thomas Gillis Wynne was born in 1891, and died after a tedious illness in March, 1959. He was educated at Geelong College, and he began the medical course in 1911. His studies were interrupted by his departure from Australia in October, 1914, with the A.I.F. Expeditionary Forces. He took no part in the landing at Gallipoli, but worked in the ships which carried the wounded from the Peninsula to the base hospitals. He was sent back to Australia in 1916 to complete his medical studies, and he graduated in 1918.

After a term as resident medical officer at the Alfred Hospital, Melbourne, he went to England in 1921 to do post-graduate work. Having no definite plans for his future career, he studied for and obtained the diploma in tropical medicine and hygiene in London. He then decided that he would specialize in ear, nose and throat work, and went to Edinburgh, where he obtained the Fellowship of the Royal College of Surgeons in 1924. Later in the same year, in London, he obtained the diploma in laryngology and otology. He became a Fellow of the Royal Australasian College of Surgeons in 1935.

After much post-graduate work at the Edinburgh Royal Infirmary, and subsequently in Vienna, he returned to Australia and commenced practice in 1925. He was appointed honorary surgeon to the ear, nose and throat department at the Alfred Hospital, and also was associated with the Austin Hospital and the Royal Children's Hospital. He inherited a comfortable legacy from his father, who was a successful medical practitioner at Colac, and this may have been a factor in delaying the development of his own practice. During the financial depression he found it necessary to go into general practice at Ripponlea. He subsequently resumed his speciality, joining the late Bryan Foster, with whom he practised for a short time as a partner. His own practice was never very large. He had a natural diffidence and reluctance to express his views emphatically, which gave some patients the impression that he was not quite sure of his opinion; but he was shrewd, painstaking and conscientious, and most of his patients became loyal friends.

He was a very active club man, belonging to the Melbourne Club, the Australian Club, the Royal Melbourne Golf Club and the principal racing clubs. His standard of billiards and golf was above the average. He was of pleasant jovial nature, and was well loved by a large circle of friends inside and outside the profession. Early in his career he was a regular attendant at medical meetings, but rarely took any active part in the discussions. He was of most exact and punctual habits, fastidious about his appearance, always dapper and well dressed. He is survived by a widow and a son.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes etc. are published in the *Commonwealth of Australia Gazette*, No. 34, of June 11, 1959.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps (Medical).

To be Temporary Major, 4th May, 1959.—2/40188 Captain F. N. Dwyer.

Regular Army Special Reserve.

Royal Australian Army Medical Corps (Medical).

NX700493 Lieutenant P. A. N. Lennon is appointed from the Citizen Military Forces, Sydney University Regiment, and to be Lieutenant, 1st January, 1959. (In lieu of the notification respecting this officer which appeared in Executive Minute No. 29 of 1959 promulgated in *Commonwealth of Australia Gazette*, No. 18, of 1959.)

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—To be Captain (provisionally), 1st May, 1959—1/39240 Douglas Forsyth Burns. *To be Lieutenant-Colonel, 28th May, 1958*—1/39206 Major (Temporary Lieutenant-Colonel) W. D. Exton.

Southern Command.

Royal Australian Army Medical Corps (Medical).—3/50291 Major D. R. Leslie is appointed from the Reserve of Officers, is appointed Consultant Surgeon, Army Headquarters, and to be Lieutenant Colonel (Temporary Colonel), 27th April, 1959. The provisional rank of 3/129438 Captain B. D. Appleby is confirmed. The resignation of 3/159573 Captain (provisionally) A. P. Ahern of his commission is accepted, 11th March, 1959.

Central Command.

Royal Australian Army Medical Corps (Medical).—4/31955 Captain J. G. Sweeney ceases to be seconded whilst in the United Kingdom, 22nd April, 1959. *To be Temporary Major, 2nd April, 1959*—4/32036 Captain T. H. Beare.

Western Command.

Royal Australian Army Medical Corps (Medical).—To be Captains (provisionally), 1st May, 1959—5/38112 Leonard Stephen James Miocevich, 5/38113 John Manneks Hill and 5/21759 Martin John Williams. *To be Temporary Major, 1st May, 1959*—5/26531 Captain M. N. I. Walters.

The following appointments, changes etc. are published in the *Commonwealth of Australia Gazette*, No. 37, of June 25, 1959.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Confirmation in Rank.—Surgeon Lieutenants (for Short Service) (on probation) Nickel Crombie, William Russell Gibson, John Ward Little, Robin Owen Stroud Sims and Alexander Scott Wilson are confirmed in the rank of Surgeon Lieutenant (for Short Service), with seniority in rank of 25th March, 1958, dated 25th March, 1959. Surgeon Lieutenant (for Short Service) (on probation) Michael John Waterfield is confirmed in the rank of Surgeon Lieutenant (for Short Service), with seniority in rank of 22nd April, 1958, dated 22nd April, 1959.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Promotions.—Surgeon Lieutenant Kenneth Frank Shepherd is promoted to the rank of Surgeon Lieutenant-Commander, dated 19th March, 1959.

Notes and News.

Foulerton Gift Research Fellowship.

Applications are invited by the Council of the Royal Society for a Foulerton Gift Research Fellowship for original research in medicine, the improvement of the treatment of disease and the relief of human suffering. The Fellowship will be tenable at any hospital, medical school or other appropriate research institution or university department in the British Isles or at any other place approved by the Council of the Royal Society. Candidates, who must be of British nationality, should supply the usual personal details and give the names of two referees. Testimonials will not be considered. The subject of the proposed research and the place at which it would be carried out, together with the name of the Head of the Department whose consent should first be obtained, must be stated.

The appointment, which is in the category of the Society's research appointments which are intended for

young persons who show exceptional promise in research, is tenable for two years in the first instance from January 1, 1960, and may be renewed annually up to a total of four years. The stipend will be in the range from £850 to £1050, depending upon qualifications. There will be superannuation benefits to which the successful candidate will be required to contribute 5% of annual stipend and to which the Society will contribute a sum equivalent to 10% of annual stipend. Children's allowances are payable.

It is a condition of the Fellowship that the successful candidate must show, if called upon to do so, that he or she is, and that his or her father and paternal grandfather are, or were at the date of their respective deaths, of British nationality. Preference will be given to candidates under the age of 35 years.

Application should be made on forms to be obtained from the Assistant Secretary, The Royal Society, Burlington House, London, W.1, and should be received as early as possible, in any case not later than October 1, 1959.

Melbourne University Regiment Association Dinner.

The Melbourne University Regiment Association at its dinner in the Melbourne University Union on Friday, September 11, 1959, will have as guest of honour Brigadier W. W. S. Johnston. Members of the profession who served with the Melbourne University Regiment are invited to attend to honour so distinguished a soldier, physician and citizen. Further particulars may be obtained from the President, Dr. W. W. Lempriere, or from the Hon. Secretary, Mr. R. T. Unkles, 162 Doncaster Road, North Balwyn, Victoria.

University Intelligence.

UNIVERSITY OF WESTERN AUSTRALIA.

DR. IAN CALDER LEWIS has been appointed Senior Assistant in the Department of Child Health of the University of Western Australia. Dr. Lewis is an Australian who was educated in London at St. Paul's School and at the University of Edinburgh, where he graduated M.B., Ch.B. in 1946 and M.D. in 1956. Apart from two years as a medical officer in the Royal Air Force, Dr. Lewis has been engaged in the study of child health and disease for the past twelve years, part of this time being spent as Tutor and Research Fellow in the Department of Child Life and Health, University of Edinburgh. Dr. Lewis returned to Australia in 1956 and has been at the Princess Margaret Hospital for Children, Perth. During the past year he has been part-time Acting Senior Assistant in the Department of Child Health.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales, in accordance with the provisions of the *Medical Practitioners Act*, 1938-59.

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (a) of the Act: Cher, Ivan, M.B., Ch.B., 1951 (Univ. New Zealand), D.O. (London), 1957, F.R.C.S (Edinburgh), 1958, F.R.C.S. (England), 1958; Nommensen, Clement Frederick, M.B., B.S., 1954 (Univ. Queensland); *Robertson, Hendry, M.B., Ch.B., 1956 (Univ. New Zealand)*; Russell, Walter McRae, M.B., Ch.B., 1918 (Univ. Melbourne); Finlay-Jones, Charles, M.B., B.S., 1959 (Univ. Sydney); *Fleming, Janet Allene, M.B., B.S., 1959 (Univ. Sydney)*; McDonald, Barbara Alexandra, M.B., B.S., 1959 (Univ. Sydney); Manion, Kevin, M.B., B.S., 1959 (Univ. Sydney); *Pannikote, Victor Malcolm, M.B., B.S., 1959 (Univ. Sydney)*; Piper, Brian James, M.B., B.S., 1959 (Univ. Sydney); Teychenné, John Lever, M.B., B.S., 1959 (Univ. Sydney); Wong, Poh Yen, M.B., B.S., *1959 (Univ. Sydney)*; *Yule*, Graham Andrew, M.B., B.S., 1959 (Univ. Sydney).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) *(b)* of the Act: Fook, William Lai, M.B.,

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 25, 1959.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	4(2)	..	..	..	..	..	..	4
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	2	..	..	..	11	..	13
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	1	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	1(1)	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	2(1)	25(24)	3(2)	..	..	..	..	..	30
Diphtheria	1	..	2	..	2(1)	..	..	..	5
Dysentery (Bacillary)	..	1(1)	3	1	6(4)	..	1	..	12
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	40(8)	20(8)	24(3)	6(2)	2(2)	..	..	..	92
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	5	..	..	..	..	..	5
Malaria	..	..	..	..	..	..	1	..	1
Meningococcal Infection	5(5)	2(2)	4	..	..	1	..	..	12
Ophthalmia	..	..	..	..	..	..	..	2	2
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	3(2)	..	..	1	..	..	..	3
Puerperal Fever	..	..	1	..	1	..	..	..	2
Rubella	..	11(10)	..	..	..	..	..	..	11
Salmonella Infection	..	..	..	..	3(3)	..	..	..	3
Scarlet Fever	8(2)	26(21)	2(2)	8(4)	1(1)	..	..	..	45
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	2(1)	..	..	..	..	..	2
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	..	27(20)	5	5(4)	10(7)	3(1)	..	..	50
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	1	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

B.S., 1934 (Univ. Hong Kong); Little, John Ward, M.B., Ch.B., 1957 (Univ. Glasgow); Peck, John Portway Seville, M.R.C.S. (England), L.R.C.P. (London), 1933, B.Chir., 1934 (Univ. Cambridge), M.B., 1936 (Univ. Cambridge), M.A., 1936 (Univ. Cambridge); Upton, John, M.B., Ch.B., 1955 (Univ. Edinburgh); Waga, Witold Jerzy, M.B., Ch.B., 1942 (Univ. Edinburgh); Waspe, Douglas Farquhar, M.B., B.Chir., 1946 (Univ. Witwatersrand).

Registered medical practitioner who has complied with the requirements of Section 17 (3) and is registered under Section 17 (2A) of the Act: Muller, Kurt, M.D., 1938 (Univ. Vienna).

The following have been issued with licences in accordance with the provisions of Section 21C of the Act: Vertes, Tibor, Division of Psychiatric Services, issued June 18, 1959; Kowalski, Ignacy, Nepean District Hospital, issued June 17, 1959.

QUEENSLAND.

The following has been registered, pursuant to the provisions of Section 19 (1) (a) and (c) of *The Medical Acts*, 1939 to 1955, of Queensland: English, Peter Francis James Devarney, M.B., B.S., 1957 (Univ. Queensland).

The following have been registered, pursuant to the provisions of Section 19 (1) (a) and (d) of *The Medical Acts*, 1939 to 1955, of Queensland: Mason, Annabel Jane Maud, M.B., B.S., 1955 (Univ. London), M.R.C.S. (England), 1955, L.R.C.P. (London), 1955; Pettit, Kenneth Edward, M.B., B.S., 1954 (Univ. Melbourne); McCutcheon, George Buchanan, M.B., Ch.B., 1952 (Univ. Aberdeen).

The following additional qualifications have been registered: Bendeich, Geoffrey Joseph, F.R.C.S., Edinburgh, 1956, F.R.C.S., England, 1957; Campbell, Colin Alexander Kemp, D.O., 1952 (Univ. Melbourne); Francis, John Brenton, D.D.M., 1959 (Univ. Sydney).

Post-Graduate Work.

SURGICAL SEMINARS AT ROYAL NORTH SHORE HOSPITAL OF SYDNEY.

THE next monthly surgical seminars will be held at Royal North Shore Hospital at 4.15 p.m., in the Students' Lecture Room, as follows: Monday, September 11, 1959, "Testicular Tumours", Dr. C. Graham, Mr. N. Wyndham. Monday, October 12, 1959, "Internal Fixation of Fractures", Mr. B. Bloch, Mr. J. Ellis, Mr. R. McGlynn.

All members of the medical profession are welcome to attend.

Corrigendum.

STUDIES ON THE USE OF ENHANCING AGENTS TO PROMOTE ABSORPTION OF TETRACYCLINE ADMINISTERED ORALLY.

WE are asked to point out that in the paper entitled "Studies on the Use of Enhancing Agents to Promote Absorption of Tetracycline Administered Orally", by Monica Bullen, Judith A. Rheuben and E. F. Thomson, which appeared in our issue of August 22, 1959, the legend to Figure I is inaccurate in two respects: the second line should read "'Tetracyn V' (sodium hexametaphosphate)"; the last line should read "'Tetracyn' + citric acid".

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Miller, Douglas Alfred Frank, M.B., B.S., 1958 (Univ. Sydney), 250 Carlingford Road, Carlingford.

Gillespie, Ailsa Margaret, M.B., B.S., 1958 (Univ. Sydney), 28 Brightmore Street, Cremorne.

Becker, Andreas Aloysius, M.D., 1940 (Univ. Szeged), (Registered under Section 17 (2B)), Public Health Department, Lae, New Guinea.

Deaths.

THE following death has been announced:

HOGG.—Geoffrey Charles Huxtable Hogg, on August 14, 1959, at Coorparoo, Brisbane.

Diary for the Month.

SEPTEMBER 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPTEMBER 2.—Western Australian Branch, B.M.A.: Branch Council.
SEPTEMBER 2.—Victorian Branch, B.M.A.: Branch Meeting.
SEPTEMBER 3.—South Australian Branch, B.M.A.: Council Meeting.
SEPTEMBER 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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